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DATABASE ASSESSMENT OF THE HEALTH AND ENVIRONMENTAL EFFECTS OF MUNITION PRODUCTION WASTE PRODUCTS

Michael C. Ryon
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August 1984

SUPPORTED BY

U.S. ARMY MEDICAL RESEARCH
AND DEVELOPMENT COMMAND
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RDX	Environmental Fate	
HMX	Manufacturing Processes	

20. ABSTRACT (Continue on reverse side if necessary and identify by block number)

A database on the health and environmental aspects of munition production waste products is presented. Information on production processes and waste treatment methods is also presented.

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FINAL REPORT

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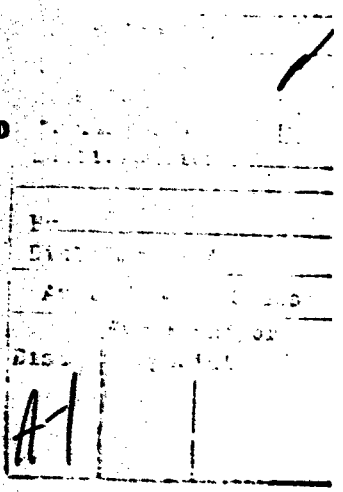

SUPPORTED BY

U.S. ARMY MEDICAL RESEARCH AND DEVELOPMENT COMMAND
Fort Detrick, Frederick, MD, under
Interagency Agreement No. 83PP3802

Project Officers: Jesse J. Barkley,
Katheryn F. Kenyon, and Allan B. Rosencrance

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EXECUTIVE SUMMARY

The modernization program launched by the Army in the early 1970s culminated in the adoption of the continuous process of manufacturing TNT to replace the batch process method. The continuous process includes the 'sellite' (sodium sulfite) purification of crude TNT, which is necessary to minimize exudation. However, the sellite treatment creates a waste product known as red water. Superior results have been claimed for a British process (ROF process) in which, during the purification step, crude TNT is contacted in the solid state with sellite rather than in the liquid state, as used in the continuous process (adopted for use in the United States). This is reported to result in a purer TNT and requires only two-thirds as much sodium sulfite. The current U.S. methods of manufacturing RDX and HMX appear to be satisfactory but can generate appreciable quantities of waste products. The yields of RDX and HMX are 80-84 percent and 55-60 percent, respectively.

It is national policy that all munition plants be government owned. Most of these plants are operated by contractors, typically a division of a major U.S. chemical company. Virtually all of the high explosives and most of the propellants for all three services are manufactured in Army Ammunition Plants (AAPs). As of 1976, the Army had 17 operational ammunition plants and one under construction in Mississippi. Of these, seven are engaged in manufacture; eight in load, assemble, and pack (LAP) operations; and two in both manufacture and LAP operations.

At the AAPs, TNT is produced in greater amounts than both RDX and HMX combined. For every 50 million pounds of crude TNT produced, 211 million pounds of raw materials (excluding water in 60 percent nitric acid) are used. Statistics on the production of the waste products red water and pink water resulting from TNT manufacture from the AAPs are not readily available. At present, the only AAP to manufacture RDX and HMX is the Holston plant in Tennessee.

The by-products formed during the manufacture of TNT and its purification by the sellite process have been identified. The maximum nominal concentration of impurities present in crude TNT is approximately 7.53 percent. This is reduced to 3.24 percent by the sellite purification step. However, of this 3.24 percent of impurities, about 1 percent were not originally present in the crude TNT, but were created during sellite treatment.

Two of the principal gaseous pollutants arising from the manufacture of TNT are nitrogen oxides (NO_x) and tetranitromethane. However, the aqueous pollutants are of greatest concern in TNT manufacture and LAP operations and are known as red water and pink water.

During the Radford continuous-process mixed-acid nitration of toluene to make TNT, the approximate maximum concentration of by-products formed is 7.5 percent. This is reduced to 2.4 percent by treatment with sellite (sodium sulfite-sodium carbonate solution), which extracts these impurities from the crude TNT. This brick-red extract is known as red water or spent sellite. The two principal organic

compounds in red water are 2,4-dinitrotoluene-5-sulfonic acid and 2,4-dinitrotoluene-3-sulfonic acid.

Pink waters are aqueous effluents from plants that manufacture or process TNT. They result from TNT contact with plant clean-up and scrubber water during manufacturing and LAP operations and as a condensate from red water evaporation concentration and incineration. The pink color is due to the photodegradation products of TNT and is visible even at initial TNT concentrations as low as 0.5 ppm. These pink constituents are a group of ill-defined, water-soluble (and organic-insoluble) anions, some displaying free-radical characteristics, which appear to constitute the major proportion of the products of TNT photolysis. In addition to TNT, other constituents initially present in the wastewaters remain after the pink color becomes manifest with the concomitant reduction in TNT. These constituents vary with the source. The most important, from a toxicological point of view, appear to be dinitrotoluene (DNT) isomers, especially 2,4-DNT and 2,6-DNT.

Although the mechanism of TNT metabolism is unknown, it is generally accepted that TNT is rapidly detoxified in the liver and excreted primarily by the kidneys. Only small amounts of the compound or its metabolites are distributed to the organs. The type and quantity of metabolites identified in the urine of TNT workers and of experimental animals receiving TNT by several routes indicate that TNT is metabolized primarily by reduction of the nitro groups and, to a lesser extent, by oxidation of the methyl group and ring hydroxylation. Metabolites have been identified from several sources, and routes of metabolism have been proposed.

RDX is extensively metabolized and does not accumulate in any tissue. After oral dosing in rats, less than 10 percent of the dose remained in the body after 4 days. It is generally concluded that the reactions involved in RDX metabolism are catalyzed by microsomal enzyme systems and occur primarily in the liver. Metabolism of RDX produces several one-carbon fragments; no larger intermediates have been identified.

No information on the metabolism of HMX, TNT isomers, tetranitromethane, trinitromethane, or methyl nitrate was available from the published literature.

Dinitrotoluene compounds (in mammals) are well absorbed after oral dosing of mammals, extensively metabolized in the liver, and rapidly excreted in the urine and, to a lesser extent, in the bile. Radioactivity of ^{14}C-labeled compounds is widely distributed in the carcass but concentrates only in the liver and kidney. Very little of the parent compounds are found in the urine. The urinary metabolites derived from 2,4-DNT, the major component of red water distillate, have been studied in detail following single-dose and chronic feeding studies, and 16 possible metabolic products have been identified. For all DNT compounds, negligible amounts of radioactivity were expired as CO_2, indicating that the aromatic ring remained intact.

A review of the literature on the toxicity of TNT, RDX, and HMX indicates that these compounds are only moderately acutely toxic. LD_{50} values for the three compounds, when administered orally to the rat, were approximately 1000, 100, and >100 mg/kg, respectively. Acute exposures with toxic reactions in humans have occurred, primarily when production rates were high during World Wars I and II and when less emphasis was placed on industrial hygiene procedures. Deaths and health effects such as hematologic changes and convulsions have been correlated with exposure levels for TNT and RDX, and standards for industrial exposures have been set. Available data indicate that HMX is less toxic than TNT and RDX. Of the waste constituents and by-products of munition production, tetranitromethane (TNM) and, to a lesser extent, nitrogen oxides have the potential to produce acute toxic effects. Workplace standards for TNM and NO_x have been set.

Although the potential for adverse health effects as a result of acute exposures is low, chronic exposures to these compounds are possible in the manufacturing environment where fumes and dusts may be generated and in areas near production sites where the compounds may be discharged to local waterways or leach into potable groundwater. In addition to munition compounds, the wastewaters contain organic and inorganic by-products for which there is limited toxicological data. The possible long-term hazards to the general population of low levels of these munition compounds and their associated by-products are of concern. Of primary concern are the long-term effects on mammalian systems from the ingestion of compounds in LAP wastewater and condensate wastewater. At low levels many of these compounds and their metabolites are mutagenic (TNT isomers and metabolites, DNT isomers and metabolites, and TNM) and carcinogenic (2,4-DNT and 2,4-diaminotoluene). When administered chronically, TNT and 2,4-DNT were associated with adverse reproductive effects in rats. In toxicity tests of LAP wastewater on mammals, there was no evidence of synergism between TNT, the principal constituent of LAP wastewaters, and RDX. Studies on other compound interactions using mammalian systems are lacking. Additional research aimed at setting standards for these compounds in drinking water is needed.

In reviewing the information on environmental effects of TNT, RDX, HMX, and associated pollutants, an impression is gained that although the short-term impact has been minimal, the potential for a greater long-term impact exists. Data on releases from AAPs indicate that aquatic systems receive the majority of the wastes. As they leave the plants, the effluents have a significant potential for harmful effects. Levels of munitions have been reported of up to 200 ppm in AAP effluents and 20 ppm in aquatic sediments. Studies on the fate and movement of TNT and RDX in aquatic systems indicate that long distance transportation of the munitions and their degradation products is not highly probable. Photolytic degradation of the compounds proceeds fairly rapidly and limits the persistence of the munitions in the water column. However, microbial degradation does not proceed rapidly, especially for RDX, and longer persistence in sediments is a distinct possibility. Studies on bioaccumulation indicate that this is not a problem for the munition compounds of concern. The impact of AAP effluents on water

quality has been demonstrated, with changes in chemical oxygen demand, concentrations of solids, nitrogen-species, phosphates, sulfates, and pH levels. These effects are usually restricted to areas immediately downstream from the AAPs, but may be quite persistent in sediments. Studies of the toxic effects of TNT and its degradation products to aquatic organisms indicate a potential for problems in the environment. Levels found to be toxic to bacteria, plants, and invertebrates in terms of growth suppression or habitat utilization (~100 ppb to 1 ppm) have been reported for both water and sediment. Levels that actually exceed LC_{50} values have also been reported for some sediment samples and for effluents from the AAPs and indicate a further hazard for certain species of bacteria, algae, vascular plants, invertebrates, and fish. Because the effective levels are higher for RDX and HMX than for TNT, the concern for potential toxic effects should be less.

The potential for deleterious effects from atmospheric releases of TNT, RDX, HMX, and associated compounds is related to the various constituent compounds arising from the manufacture of the munitions. The principal species of concern are sulfur dioxide, nitrogen oxides, and particulates. These have been found to exceed the allowable EPA levels for release from AAPs, in terms of both daily and yearly amounts. The small amount of information available on transportation and persistence suggests that the problems associated with the releases should be local in nature. Studies of the toxic effects of these releases are lacking, but the information available indicates that the sulfur dioxide releases pose a potential problem for surrounding vegetation.

Data on the impact of TNT, HMX, and RDX on the soils, groundwater, and associated terrestrial ecosytems are generally insufficient for in-depth analysis. Data available on the input of munitions to terrestrial systems suggest that high levels could be present in limited areas. Because other information indicates that the munitions may be persistent at depths, due to slow biodegradation, some concern is merited regarding potential deleterious effects. Migration studies in soil do indicate that the munitions move slowly, producing a gradient of decreasing concentrations which should limit the immediate impact to local areas. Chronic groundwater contamination is an associated potential hazard from terrestrial releases. The most likely source for problems of these types would be improper disposal in landfills.

Most of the available information on the release of munition wastes to the environment came from documents published in the mid to late 1970s; documentation is not readily available to indicate if pollution abatement and waste treatment methodologies have been updated in respective AAPs and LAP facilities.

In modernized AAPs, air pollution does not appear to be a significant problem, and explosive solid wastes will undergo controlled incineration. Nitrogen oxides and tetranitromethane are removed effectively by a two-stage scrubbing with sulfuric acid in conjunction with another two-stage scrubbing with sellite.

Red water and pink water are the major sources of potential water pollution. The SONOCO sulfite recovery process is claimed to greatly reduce the waste handling problems associated with red water. The current status of this process with respect to use in AAPs has not been reported. Pink water is treated by activated charcoal. A method has been developed for the regeneration of the explosive-laden charcoal with 92 percent efficiency, thus considerably reducing the operation costs.

During munitions manufacture heavy metals are apparently leached from the nitrating vessels as a result of the action of the nitrating acid mixture. Attempts to remove these heavy metals from the wastewaters have not been reported.

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1. INTRODUCTION

The purpose of this review is to provide information to the U.S. Army Medical Research and Development Command on several aspects of the waste problem connected with the manufacture of the munitions 2,4,6-trinitrotoluene or alpha-TNT, hexahydro-1,3,5-trinitro-1,3,5-triazine (RDX), and octahydro-1,3,5,7-tetranitro-1,3,5,7-tetrazocine (HMX). The initial objectives included: identifying those munition production wastes having the greatest potential for causing adverse health and environmental effects; providing the Army with a data base that will support future decision-making processes concerning additional delistings from RCRA requirements; and beginning the examination of the rationale for the selection of efficient methods of disposal of munition production wastes.

To accomplish these objectives, computer searches were performed of online bibliographic data bases including MEDLINE, TOXLINE, Chemical Abstracts, Biological Abstracts, and the holdings of the Defense Technical Information Center (DTIC). Hardcopies of pertinent references were obtained and used in compiling the review. Manual searching of reference citations in the hardcopy documents provided additional literature for the report. Based on the data available in the literature, the document was divided into sections covering the following: the physical and chemical properties of the primary munition compounds and some associated compounds, the manufacturing processes and production statistics for the Army ammunition plants, the metabolism and health effects in humans and test animals, the environmental fate and effects, and current and experimental waste treatment procedures. The format for the discussions is basically text and tables, but due to ease of presentation the section on physical and chemical properties (Section 2) is arranged in an outline format. Discussed throughout the document are the data gaps and research needs identified as a result of the analysis of the literature. The compounds, especially TNT, are designated in this text based on how they are identified in the cited literature; if the reference specifies alpha-TNT, then we also cite it as alpha-TNT. The general term TNT is used where the cited reference is nonspecific.

2. PHYSICAL AND CHEMICAL PROPERTIES OF TNT, RDX, HMX, AND SELECTED WASTE PRODUCTS ASSOCIATED WITH THEIR PRODUCTION

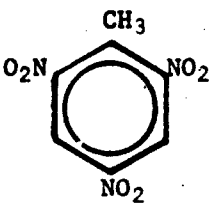
2.1 TNT

Data from Lindner 1980, unless indicated otherwise.

Molecular formula: $C_7H_5N_3O_6$

Molecular weight: 227.13

Structural formula:



CAS registry number: 118-96-7

Chemical name: 2,4,6-Trinitrotoluene

Synonyms: TNT, 2-methyl-1,3,5-trinitrobenzene, α-trinitrotoluol, sym-trinitrotoluene, 1-methyl-2,4,6-trinitrobenzene, Tolit, Trilit, Trotyl, tritol, sym-trinitrotoluol (Windholz 1976)

Elemental analysis: C, 37.01 percent; H, 2.22 percent; N, 18.50 percent; O, 42.27 percent (Windholz 1976)

Melting point: 80.1°C (Windholz 1976)

Density: D_4^{20} 1.654 (solid) (Windholz 1976)

$d(g/cm^3) = 1.5446 - 1.016 \times 10^{-3}t$, where t = °C; applicable in the temperature range 83°-120°

Dipole moment: 1.37D (Windholz 1976)

Hygroscopicity: Nonhygroscopic

Vapor pressure: 0.046 mm Hg at 82°C (TDB 1982)

Stability: α-2,4,6-Trinitrotoluene is very stable and may be stored indefinitely at temperate conditions without deterioration.

Decomposition: The decomposition mechanism of TNT at elevated temperatures (200°C) is very complex, producing at least 25 different compounds as well as large amounts of undefined polymeric material.

Viscosity: 8 cP at 99°C

Detonation products (experimentally determined in a calorimetric bomb, mole per mole TNT): 3.65 C (sol), 1.98 CO, 1.60 H_2O, 1.32 N_2, 0.46 H_2, 0.16 NH_3, and 0.10 CH_4

Heat of formation, kJ/g: 0.293

Heat of combustion, kJ/g: 15.02

Heat of detonation, kJ/g: 4.23

Specific heat, J/(g·K): 1.38

Heat of fusion, J/g: 98.3

Heat of vaporization, J/g: 339

Heat of sublimation, J/g: 447

Thermal conductivity, W/(m·K): 0.54

Coefficient of linear expansion: 6.7×10^{-3}/°C

Solubility (g/100 g) at 20°C (Windholz 1976):

Water: 0.01

Other solvents:

Pyridine - 137	1,2-Dichloroethane - 18.7
Acetone - 109	Diethyl ether - 3.29
Methyl acetate - 7211	Trichloromethylene - 3.04
Benzene - 67	95 percent ethanol - 1.23
Toluene - 55	Carbon tetrachloride - 0.65
Chlorobenzene - 33.9	Carbon disulfide - 0.48
Chloroform - 19	

Specifications for two military grades of TNT (Gilbert 1980):

Property	General Type I	Special Type II
Solidification point (°C-min)	80.2	80.4
Moisture, % max	0.10	0.10
Acidity (as H_2SO_4), % max	0.02	0.02
Alkalinity	None	None
Insoluble matter, % max	0.05	0.05
Sodium, % max	0.001	0.001
Color	Light yellow through buff	Light yellow

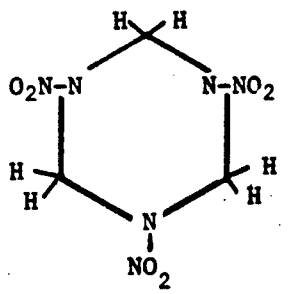
2.2 RDX

Data from Lindner 1980, unless indicated otherwise.

Molecular formula: $C_3H_6N_6O_6$

Molecular weight: 222.13

Structural formula:



CAS registry number: 121-82-4

Chemical name: Hexahydro-1,3,5-trinitro-1,3,5-triazine

Synonyms: RDX (British code name for Research Department Explosive or Royal Demolition Explosive, cyclotrimethylene trinitramine, cyclonite, hexogen, PBX (AF) 108, T4, 1,3,5-triaza-1,3,5-trinitrocyclohexane, trimethylenetrinitramine, 1,3,5-trinitroperhydro-1,3,5-triazine

Elemental analysis: C, 16.22 percent; H, 2.72 percent; N, 37.83 percent; O, 43.22 percent

Color: White

Crystal density, g/cm^3: 1.83

Crystal form: Orthorhombic

Melting point: 204°C

Hardness, Mohs: 2.5

Oxygen balance, percent to CO_2: -22

Heat of formation, kJ/g: -0.277

Heat of fusion at 478.5 K: 8.5 kcal/mole (Roth 1980)

Heat of combustion, kJ/g: 9.46

Specific heat, J(g°K): 1.26; 0.398 cal/g°C at 20°C (Roth 1980)

Heat of vaporization, J/g: 490

Heat of sublimation, kcal/mole: 31.1 (Roth 1980)

Detonation products (calculated values, mole per mole RDX): 3.00 N_2, 3.00 H_2O, 1.49 CO_2, and 0.02 CO

Stability: Stored at 85°C for 10 months without perceptible deterioration.

Dipole moment: ~7D (in highly polar solvents) (Roth 1980)

Solubility (g/100 g) at 20°C: (Gilbert 1980)

Solvent	25°C	40°C	60°C	80°C	98°C
Dimethyl sulfoxide	41	51	66	87	113
Dimethyl formamide	37	45	58	76	96
N-Methylpyrrolidone	40	47	58	72	84
Butyrolactone	14	-	29	41	61
Acetone	8.2	12	17 (56.5°C)	-	
Butyrolactone: dimethyl sulfoxide (73:27)	26	-	33	-	49

Solubility: water at 25°C, 7.6 mg/L; water at 83°C 1.3 g/L; acetone at 30°C, 69.0 g/L; cyclohexanone at 30°C, 84.0 g/L; acetic anhydride at 30°C, 49.0 g/L (Patterson et al. 1976)

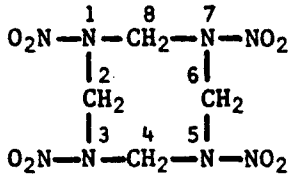
2.3 HMX

Data from Lindner 1980, unless indicated otherwise.

Molecular formula: $C_4H_8N_8O_8$

Molecular weight: 296.17

Structural formula:



CAS registry no.: 2691-41-0

Chemical Name: Cyclotetramethylenetetranitramine

Synonyms: HMX (High Melting Explosive, Fedoroff and Sheffield 1966), β-HMX homocyclonite, HW4, LX 14-0, Octahydro-1,3,5,7- tetranitro-1,3,5,7-tetrazocine, octogen, tetramethylenetetranitramine, 1,3,5,7-tetraza-1,3,5,7-tetranitrocyclooctane

Elemental analysis: C, 16.22 percent; H, 2.72 percent; N, 37.84 percent; O, 43.22 percent

Color: White

Crystal density, g/cm^3: 1.90 (beta)

Crystal form: Four polymorphic forms, beta-form least sensitive and most stable

Melting point: 286°C

Hardness, Mohs: 2.3

Solubility, g/L: Water at 83°C - 0.14; acetone at 30°C - 22.0; cyclohexanone at 30°C - 53.0; acetic anhydride at 30°C - 13.0 (Patterson et al. 1976)

Oxygen balance, percent to CO_2: -22

Heat of formation, kJ/g: -0.253

Heat of combustion, kJ/g: 9.43

Heat of detonation, kJ/g: 5.67

Specific heat, J(g.K): 1.26

Heat of vaporization, J/g: 368

Detonation products (experimentally determined in a calorimetric bomb, mole per mole HMX): 3.68 N_2; 3.18 H_2; 1.92 CO_2; 1.06 CO; 0.97 C, 0.395 NH_3; 0.30 H_2

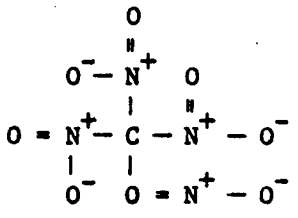
2.4 TETRANITROMETHANE

Data from Clayton and Clayton 1982, unless indicated otherwise.

Molecular formula: $C(NO_2)_4$

Molecular weight: 196.04

Structural formula:



CAS registry no.: 509-14-8

Chemical Name: Tetranitromethane

Synonyms: Tetranitromethan (Ger), Tetranitrometheno (Itl), Tetranitromethan (Rus) (McDonnell 1978)

Elemental Analysis: C, 6.13 percent; N, 28.58 percent; O, 65.29 percent

Melting point: 14.2°C

Boiling point: 125.7°C

Density: 1.62 294/25°C

Refractive index: 1.43822 (McDonnell 1978)

Solubility: Insoluble in water at 20°C

Vapor pressure: ~13 mm Hg/25°C, 8.4 torr/20°C (11,000 ppm or 90,000 mg/m^3)

Vapor density: 0.9 (air = 1)

Assay: Collection in reagent grade methanol followed by reading at 240 nm and comparison with standard calibration curves using MS, GC, or IR.

Color: Colorless, but becomes yellow on contact with water due to hydrolysis to trinitromethane.

Odor: Distinct pungent odor (characteristic acrid biting odor)

Physical state: oily fluid

Explosion temperature: Does not explode below 360°C (McDonnell 1978)

Heat of combustion: 102.9 kcal/mole (McDonnell 1978)

Heat of explosion: From differential thermal analyses, exothermic at 310°C; the Qe at 227°C was calculated to be 557 cal/g (McDonnell 1978)

Heat of formation: -8.9 kcal/mole (McDonnell 1978)

Spectrum, ultraviolet: λ_{max} ~275 nm, log ε 2.2 (McDonnell 1978)

Thermal stability: At 100°C it evolves acid fumes in 30 min, at 135°C there is no explosion after 300 min (McDonnell 1978)

Formation from TNT: By the use of ^{14}C-labeled TNT, it was shown that 54.2 percent of the tetranitromethane came from the aromatic C attached to the methyl group, 8.3 percent from each of the C atoms connected to nitro groups, 7.4 percent from each of the C atoms attached to H, and 6.1 percent from the methyl group (McConnell 1978)

2.5 TRINITROMETHANE

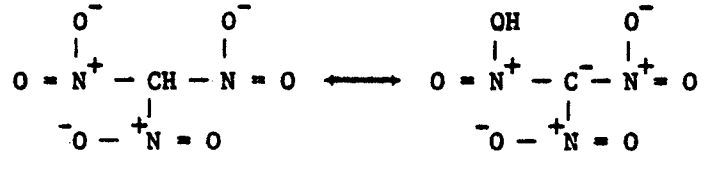
Data from McDonnell 1978.

Synonyms: Nitroform (German), Nitroforme (French)

CAS registry no.: 517-25-9

Molecular formula: $CH(NO_2)_3$

Structural formula:



Molecular weight: 151.04

Melting point: 26.3 ± 0.1, higher melting form (aci-form) melts at 50°C

Density: 1.5967 at 24.3°C

Boiling point: 45-47°C/22 mm Hg

Explosion hazard: Explodes when heated rapidly; can explode during distillation

Physical state: Colorless to pale yellow solid

Refractive index: 1.44174 at 24.3°C

Acidity: The pH of a saturated aqueous solution, 0.6

Spectrum, ultraviolet: Shows a plain shoulder in ethanol, and in water shows a broad maximum at 340 nm

Stability: May be stored indefinitely at 0°C in sealed glass ampoules

Chemical reactions: Trinitromethane is a strong acid and forms salts with metals and organic bases; it forms complexes with benzene and toluene

2.6 METHYL NITRATE

Data from Clayton and Clayton 1982.

Molecular formula: CH_3NO_3

Structural formula: CH_3ONO_2

Molecular weight: 77.042

Boiling point: 65°C (explodes)

Specific gravity: 1.217 (15°C)

Vapor density: 2.66 (air = 1)

Solubility: Slightly soluble in water, soluble in alcohol and ether

2.7 SEX

Data from Fedoroff et al. 1960 unless indicated otherwise.

Chemical name: 1-Acetyloctahydro-3,5,7-trinitro-1,3,5,7-tetrazocine

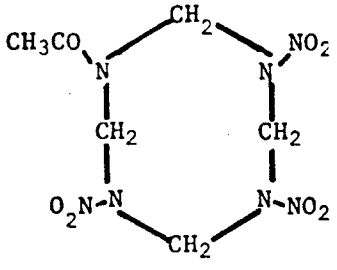
Synonyms: ODX, 1-acetyl-3,5,7-trinitro-octahydro-s-triazine, octahydro-1-acetyl- 3,5,7-trinitro-s-tetrazocine

CAS registry no.: 13980-00-2

Molecular formula: $C_6H_{11}N_7O_7$

Elemental analysis: C, 24.68 percent; H, 3.78 percent; N, 33.44 percent; O, 38.20 percent Molecular weight: 293.208

Structural formula:



Melting point: 224.2-224.7°C with frothing

Solubility: Slightly soluble in pyridine, acetone, and nitromethane; nearly insoluble in alcohol, benzene, acetic acid, and ether

Ultraviolet spectrophotometric data: λ_{max} 227nm; ε_{max}, 15,800 (Schroeder et al. 1951)

2.8 TAX

Data from Fedoroff et al. 1960 unless indicated otherwise.

Chemical name: 1-Acetylhexahydro-3,5-dinitro-1,3,5-triazine

CAS registry no.: 14168-42-4

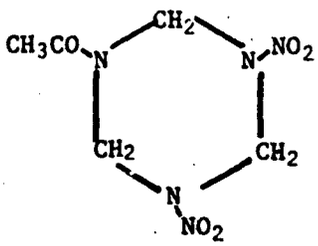
Synonyms: 1-Aceto-3,5-dinitro-1,3,5-triazacyclohexane, 1-acetyl-3,5-dinitro-s-triazine, 1,5-dinitro-3-acetyl-1,3,5-triazine

Molecular weight: 219.162

Molecular formula: $C_5H_9N_5O_5$

Elemental analysis: C, 27.4 percent; H, 4.14 percent; N, 31.96 percent; O, 36.50 percent

Structural formula:



Melting point: 156-158°C

Solubility: Soluble in acetone, alcohol

Ultraviolet spectrophotometric data: λ_{max} 231-234 nm, ε_{max} 6,500 (Schroeder et al. 1951)

3. MANUFACTURING PROCESSES FOR TNT, RDX, AND HMX

3.1 MANUFACTURE OF TRINITROLUENE (TNT)

TNT is manufactured by stepwise nitration of toluene in batch or continuous modes of operation (Gilbert 1980). The continuous process has many advantages over the batch process, including savings in space and labor, more rapid throughput, higher yields, and better product uniformity, process control, and safety. Batch operation was, however, used exclusively during World War II in the United States, and it was not until 1968 that TNT was produced by continuous operation; this occurred at the Radford Army Ammunition Plant (AAP).

A 1953 study group (Gilbert 1980, citing data of Wendes and Little 1954) studied the known European continuous processes and concluded that the Bofors Norell Process was the best. This process entails purification of TNT by crystallization from nitric acid. A second study group in 1967 selected the Swedish Nobel-Chematur Process, as modified by Canadian Industries Ltd. (CIL) (using sodium sulfite for the purification step), as the best available continuous process (Gilbert 1980, citing data of Slemrod 1970). It was a further modification of this process that became the first continuous operation of TNT manufacture in the United States in 1968 at the Radford AAP.

3.1.1 Radford Process

The Radford AAP procedure for TNT manufacture is an improved version of the original Swedish Nobel-Chematur Process as modified by CIL using sodium sulfite for the purification step (Gilbert 1980). As originally put into operation, the procedure nitrated toluene in six stages to TNT using a nitric acid-sulfuric acid mixture. The temperature and the composition of the nitrobody at each of the six stages are shown in Table 3.1.

TABLE 3.1. SIX-STAGE RADFORD AAP NITRATION PROCESS[a]

Stage	1	2	3	4	5	6
Temperature (°C)	50-55	70	80-85	90	95	100
Composition of Nitrobody (%):						
MNT	77.1	-	-	-	-	-
DNT	18.2	70.9	30.4	10.3	1.6	0.2
TNT	4.2	28.9	69.3	89.5	98.2	99.7

a. Data from Gilbert 1980.

Each stage utilizes a 500-gal nitrator and a 150-gal acid-nitrobody separator for producing 50 tons of TNT per day, except for Stages 1 and 3, which each have two nitrators and one separator (Gilbert 1980). The process is shown schematically in Figure 3.1 (Patterson et al. 1976).

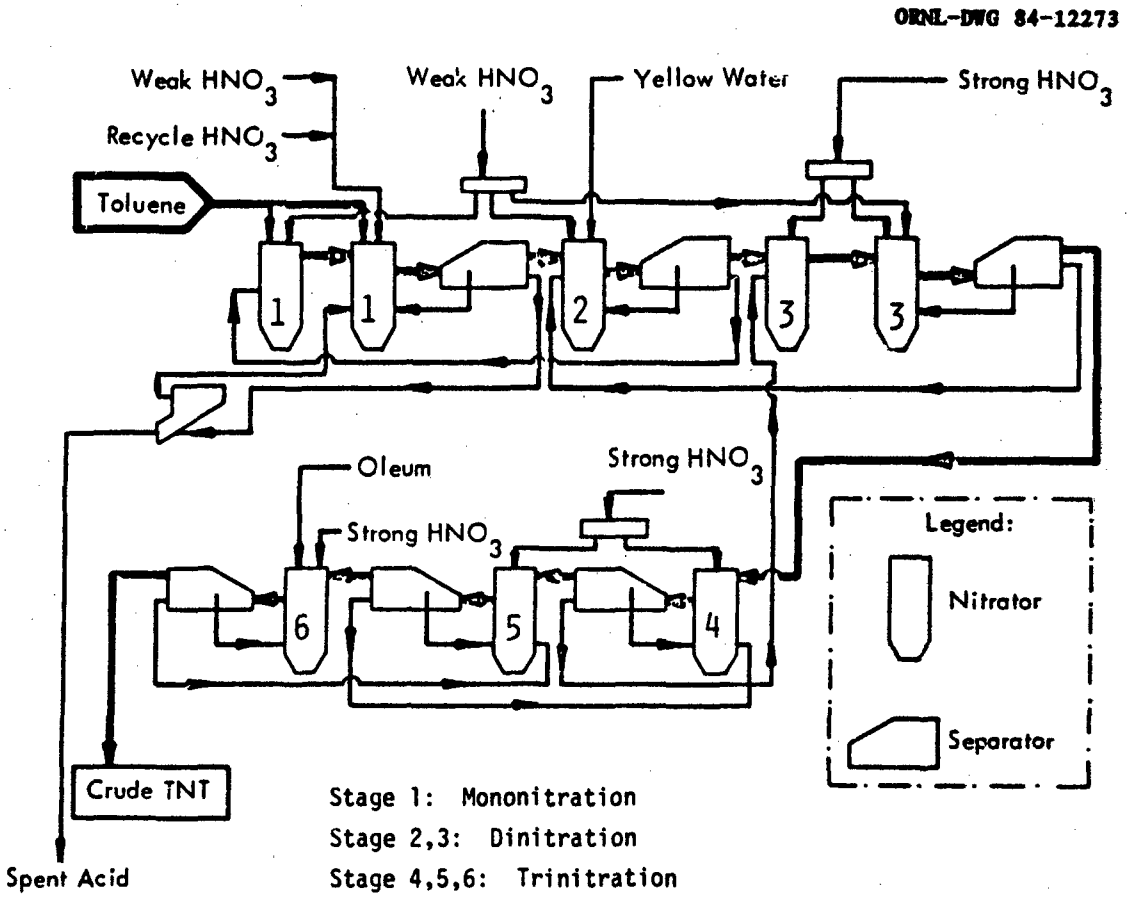


Figure 3.1. Radford AAD continuous process for TNT manufacture. From Patterson et al. 1976.

The crude TNT is subjected to sodium sulfite purification, also known as sellite purification, shown schematically in Figure 3.2. The impurities in crude TNT must be removed because their presence may lead to instability of TNT at higher temperatures and to the formation of low melting eutectics that separate over a period of time and cause problems in casting of explosive formulations (Lindner 1980). Gilbert (1980) states that for several reasons, such as a notably higher than expected consumption of raw materials, this process has undergone steady modification since its startup. An explosion in 1974, which destroyed one of the three continuous lines, also indicated a need for process modification. It was thus realized for the first time that acid-nitrobody emulsions can be explosive; previously the assumption was that only the separated nitrobody phase was potentially dangerous. To ensure maximum safe operation, the number of nitration stages was increased from six to eight. This entailed replacing the six gravity separators by eight more effective dynamic (centrifugal) separators resulting in drastic reduction of the total quantity of nitrobody present in the building at one time to 40-50 percent of that present with the use of gravity separators.

3.1.2 British Royal Ordnance Factory and West German Processes

The Royal Ordnance Factory Process (ROF) currently used in England is another continuous process for the manufacture of TNT (Gilbert 1980). This process uses 96 percent sulfuric acid, which requires more staging to effect complete nitration than the Radford process, which employs 40 percent H_2SO_4. Gilbert (1980, citing data of Bolleter 1974) states that the Radford use of sulfuric acid is about 79 percent that of the ROF process, both calculated on the basis of a strength of 96 percent. The ROF system employs a lower temperature for mononitration, 35 to 42°C, in contrast with 50 to 55°C for the Radford process. The yield of TNT in both processes is about the same, 85 percent of theoretical.

In West Germany, mononitration is achieved by a continuous process, but di- and trinitration are conducted batchwise. Trinitration is achieved with mixed acid consisting of 24 percent HNO_3, 70 percent H_2SO_4, and 6 percent SO_3, with the reaction requiring six hours with slowly rising temperatures.

3.1.3 Impurities and Purification Processes

All crude TNT is purified before military use to remove impurities which lower the melting point and cause exudation during storage by the formation of low-melting eutectics (Gilbert 1980). Gilbert (1980, citing data of Urbanski 1964) states that prior to World War I, purification of crude TNT was accomplished by recrystallization from an organic solvent, such as ethanol. After that time, it was discovered that more economical purification, which was also easier and safer, could be achieved using aqueous sodium sulfite (sellite), and this has become standard practice (see Figure 3.2).

There are six possible TNT isomers (Table 3.2). The α or 2,4,6-isomer is the isomer of military interest and is the one that is

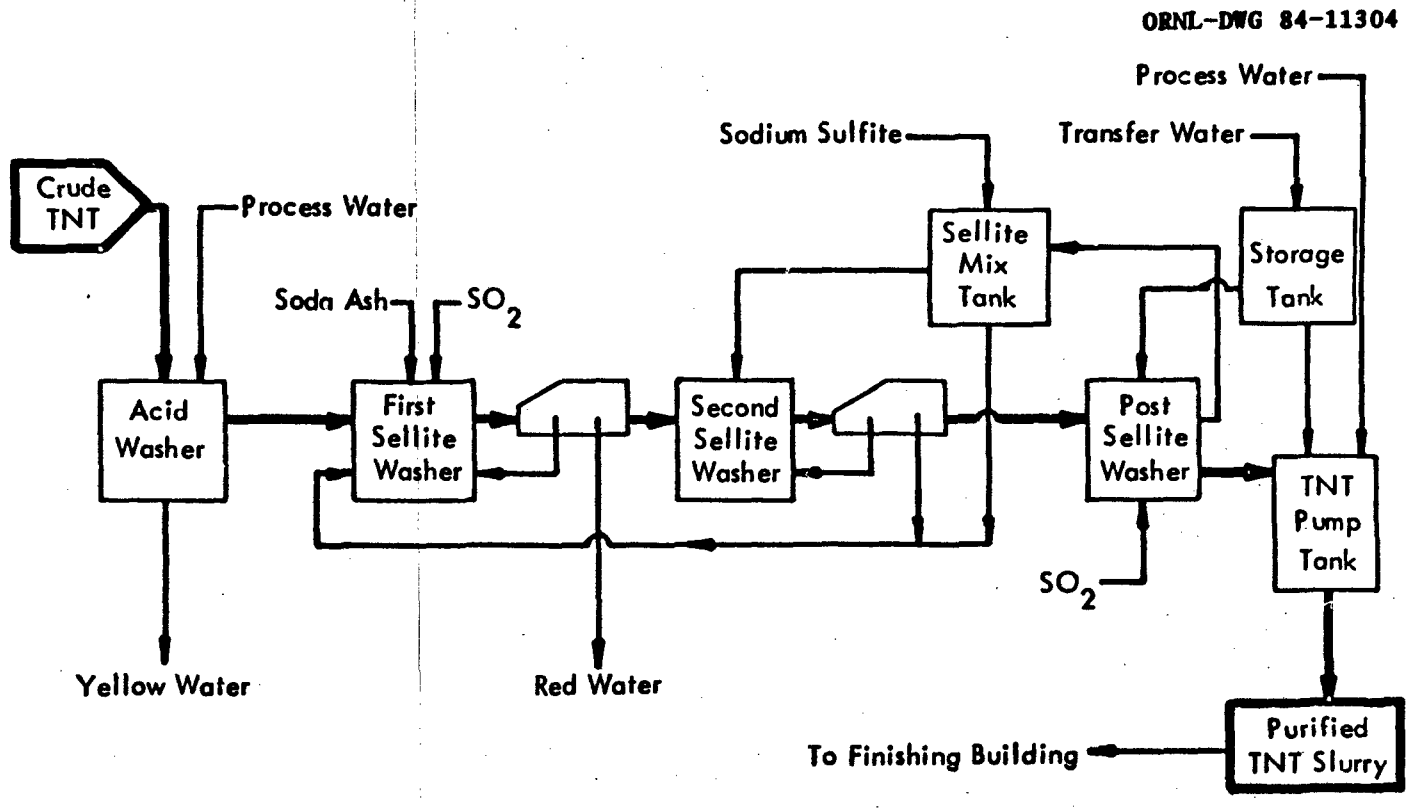


Figure 3.2. Sellite purification of TNT. From Patterson et al. 1976.

commonly designated TNT (Gilbert 1980). The other five are meta (unsymmetrical) isomers and are formed as by-products during the manufacture of TNT (see Group 1, Table 3.3). The 2,4,5-isomer is the most abundant meta isomer (also the most abundant of all impurities) in crude TNT followed by the 2,3,4-, 2,3,6-, 2,3,5-, and 3,4,5-isomers. Each of these meta isomers contains one reactive NO_2 group as shown in Table 3.2 and can react with sodium sulfite to form water-soluble sulfonates, whereas α-TNT is practically inert to this reagent, which is used for the purification of crude TNT. As shown in Table 3.3, complete removal of the meta TNT isomers by the sellite process is not achieved; about 0.6 percent remains in the purified TNT. Also, as shown in Table 3.3 (Group 2), all six possible dinitrotoluenes (DNTs) are formed, and these are not removed by the sellite purification process. These are formed as a result of incomplete trinitration and comprise about 1 percent of the finished TNT. In contrast, in the batch trinitration process (i.e., the West German process discussed in Section 3.1.2), very little of the DNT isomers is present in the reaction mixture.

TABLE 3.2. TNT ISOMERS

Isomer	Position of Reactive Nitro Group
2,4,6 (α)	none
2,3,4 (β)	3
2,4,5 (γ)	5
3,4,5 (δ)	4
2,3,5 (ε)	2
2,3,6 (η)	3 (?)

The methyl group in TNT is highly reactive and can undergo various degrees of oxidation during the manufacture of TNT. All of the compounds in Group 3 (Table 3.3) are formed directly or indirectly from oxidation of the methyl group during nitration. The formation of 2,2'-dicarboxy-3,3',5,5'-tetranitroazoxybenzene (white compound) indirectly contributed to the explosion at Radford AAP. As previously mentioned, installation of dynamic centrifugal separators in place of gravity separators solved this problem. It led to cleaner separation of the acid from the nitro-body and thus avoided the buildup of the "white compound" on the nitrator cooling coils.

The three compounds listed in Group 4 (Table 3.3) are not present in the crude TNT but are formed during the purification process. From Table 3.2, the approximate maximum concentration of impurities in crude TNT is 7.53 percent and in finished TNT is 3.24 percent.

As discussed by Gilbert (1980) the ROF TNT process also employs continuous purification, using a compartmented reactor generally similar to that used for nitration. However, the TNT is contacted as a solid at 67.5°C, rather than as a liquid at >80°C, as in the Radford process. This is said to result in a higher yield of purer TNT and requires only about two-thirds as much sodium sulfite. This procedure takes advantage

TABLE 3.3. IMPURITIES PRESENT IN TNT PREPARED BY CONTINUOUS NITRATION AND PURIFICATION[a,b]

Compound	Approximate Maximum Nominal Concentration (%)[c]	
	Crude	Finished
Group 1[d]		
2,4,5-Trinitrotoluene	2.50	0.30
2,3,4-Trinitrotoluene	1.75	0.20
2,3,6-Trinitrotoluene	0.50	0.05
2,3,5-Trinitrotoluene	0.05	0.05
Group 2		
2,6-Dinitrotoluene	0.25	0.25
2,4-Dinitrotoluene	0.50	0.50
2,3-Dinitrotoluene	0.05	0.05
2,5-Dinitrotoluene	0.10	0.10
3,4-Dinitrotoluene	0.10	0.10
3,5-Dinitrotoluene	0.01	0.01
Group 3		
1,3-Dinitrobenzene	0.02	0.02
1,3,5-Trinitrobenzene	0.15	0.10
2,4,6-Trinitrobenzyl alcohol	0.25	0.25
2,4,6-Trinitrobenzaldehyde	0.25	0.25
α-Nitrato-2,4,6-trinitrotoluene	0.10	0.10
Tetranitromethane	0.10	none
2,2'-Dicarboxy-3,3',5,5'-tetranitroazoxybenzene ("white compound")	0.35	0.05
Group 4		
2,2',4,4',6,6'-Hexanitrobibenzyl (HNBB)	none	0.40
3-Methyl-2',4,4',6,6'-pentanitrodiphenylmethane (MPDM)	none	0.40
3,3',5,5'-Tetranitroazoxybenzene	none	0.01

a. Data from Gilbert 1980.

b. Information from Radford Army Ammunition Plant, Radford, Va.

c. Concentrations listed are the maximum possible. Actual values may be much lower.

d. The 3,4,5-isomer has also been identified in crude TNT to the extent of 0.006%.

of the fact that the unsymmetrical TNT isomers form a eutectic which migrates to the crystal surface. 2,2',4,4',6,6'-Hexanitrobibenzyl (HNBB) and 3-methyl-2',4,4',6,6'-pentanitrodiphenylmethane (MPDM) are not formed. This type of process is also employed in the West German scheme (see Section 3.1.2) where mononitration is effected by a continuous process, but di- and tri-nitration are conducted batchwise because of a high purity requirement. Due to the long reaction time, this process removes nearly all of the DNT isomers yielding a TNT of unusually high purity.

Nitric acid has been used for many years in Sweden for the purification of TNT by recrystallization and has certain advantages over the sellite process previously discussed (Gilbert 1980). One advantage is that dinitro compounds are removed by nitric acid recrystallization and less drastic conditions for nitration can therefore be used to minimize oxidative losses, because nearly complete conversion of the dinitro compounds is not necessary. Nitric acid is recovered from the mother liquor by distillation and the residue, known as isotriol, consists of about 50 percent TNT, 25 percent TNT meta isomers, and 25 percent dinitrotoluenes and oxidation products. Gilbert states that, in spite of these substantial advantages, this process has not been adopted for use elsewhere because of the problem of locating reliable outlets for isotriol.

Gilbert (1980) cites several references in stating that ammonium sulfite and magnesium sulfite have shown promise in laboratory studies as alternatives to sodium sulfite in the sellite process. With ammonium sulfite, a small amount of ammonia is liberated in the process, and it reacts with the meta-TNT isomers forming dinitrotoluidines which lower the melting point of TNT. One of the advantages of this method is that the red water disposal problem (see Section 5.3) is eliminated, because the wash water contains no metallic salts and can be mixed with spent acid. Also, sulfur is recovered from the spent acid, and the ammonia is oxidized to nitrogen. The use of magnesium sulfite gives better yields of TNT of higher purity, and the magnesium is easier to recover. This can be effected in one step:

$$MgSO_4 + \tfrac{1}{2}C \rightarrow MgO + SO_2 + \tfrac{1}{2}CO_2 \ .$$

However, magnesium sulfite is less convenient to use because it has a lower solubility in water. The use of magnesium bisulfite in the paper industry has been reported (Windholz 1976) and may be worth further exploration for possible application in this area.

It has been suggested that complete purification of TNT may not be necessary in some cases, especially with modern bomb assembly and sealing techniques and the availability of additives to prevent exudation of cast TNT mixtures (Gilbert 1980). This proposal merits serious consideration as it will greatly minimize pollution and costs associated with production.

3.1.4 Exudation of TNT

As discussed by Gilbert (1980), the principal causes of exudation in TNT are the impurities present and the alcohol used for cleaning the shell threads or as an ingredient of the shellac used in the booster cavity. One current method of testing for exudation consists of tightly wrapping a 1 inch x 5 inch casting of the explosive sample with Whatman No. 1 filter paper and sealing it hermetically in a heavy-walled aluminum cylinder. The sample is then held at 71°C for 6 days. The exudate absorbed by the paper is measured. Military grade TNT typically shows 0.6 to 1.2 percent exudate, although Gilbert (1980) states that this upper limit may be higher.

A typical analysis of exudate from specification grade TNT, made at Radford AAP by the continuous process, is as follows (Gilbert 1980):

2,4,6-TNT (α)	88.19%
2,3,4-TNT (β)	1.99%
2,4,5-TNT (γ)	0.67%
2,4-DNT	7.65%
2,6-DNT	0.98%
2,5-DNT	0.19%
Hexanitrobibenzyl (HNBB) and Methylpentanitrodiphenylmethane (MPDM)	0.1 to 0.5%

All the compounds listed above form low-melting eutectics with α-TNT which exude. Two approaches have been considered for minimizing exudation (Gilbert 1980). One approach is to choose a production process to form TNT with a limited amount of impurities. The formation of dinitrotoluenes can be avoided by using more drastic nitration conditions (i.e., recrystallization of TNT from nitric acid). The formation of HNBB and MPDM can be minimized by more precise selliting at a controlled pH. The second alternative is the addition of small amounts (0.6 percent) of cellulose esters or polyurethane to remove exudate by absorption.

3.1.5 Composite Explosives

The low melting point (81°C) and thermal stability of TNT make it suitable for use as a carrier for melt-loading of more powerful explosives, such as RDX and HMX, which cannot be used alone because of their high melting points (Gilbert 1980). Loading and fabrication procedures have been described by Gilbert (1980). Of the various composites, composition B (containing 36 to 40 percent TNT and 64 to 60 percent RDX) has been the most important bomb-charge explosive.

3.2 MANUFACTURE OF RDX AND HMX

RDX, cyclotrimethylenetrinitramine, is the third most important explosive from a tonnage viewpoint after TNT and NC (nitrocellulose) (Fedoroff and Sheffield 1966).

The principle for RDX manufacture exclusively used in the United States is rather simple and is known as the Bachmann Process (Lindner 1980). Hexamine is nitrated with a mixture of nitric acid, ammonium nitrate, acetic acid, and acetic anhydride (Figure 3.3) (Fedoroff and Sheffield 1966). In this reaction, hexamine is nitrated to form one mole of RDX; three molecules of formaldehyde, liberated from hexamine, react with ammonium nitrate in the presence of acetic anhydride to form a second mole of RDX.

In this reaction, the normal yield of RDX is doubled (80 to 84 percent), compared to that obtainable by the direct nitration of hexamine with nitric acid alone, and 8 to 12 percent of HMX is formed as a by-product. The RDX formed is known as Type B in contrast to Type A, which does not contain HMX as an impurity. Many other by-products are formed in this reaction but may not always be present. Ten of these have been identified (Fedoroff and Sheffield 1966):

1. DPT or 1,5-endo-methylene-3,7-dinitro-1,3,5,7-tetrazacyclooctane
2. BSX or 1,7-diacetoxy-2,4,6-trinitro-2,4,6-triazaheptane
3. Bromo-DPT or 1,5-endo-ethylene-3,7-dinitro-1,3,5,7-tetrazacyclooctane
4. 1,5-endo-ethylidine-3,7-dinitro-1,3,5,7-tetrazacyclooctane
5. 1-nitro-3,5-dicyclohexyl-1,3,5-triazacyclohexane
6. N,N'-dimethylol-N,N'-dicyclohexyldiaminoethane
7. 3,7-dinitro-3,7-diaza-1,5-dioxacyclooctane
8. dinitroxydimethylnitramide
9. diacetoxydimethylnitramide
10. 1,3,5-trinitroso-1,3,5-triazacyclohexane

In addition to the above list, SEX and TAX have been reported to be formed during nitrolysis of hexamine (Fedoroff et al. 1960). SEX has been found to be present in the effluent wastewater from Holston AAP at levels of 2.5 ppm (Chen et al. 1981), and Stidham (1979) has reported the presence of both SEX (<2.03 mg/L) and TAX (<5.24 mg/L) in the wastewaters from Holston AAP.

In the Bachmann process, the reactants are mixed, and the slurry is aged to complete the reaction and increase the yield. It is then diluted and simmered to destroy by-products (Lindner 1980). The RDX-acetic acid slurry is filtered, water-washed, and the spent acetic acid is processed for recovery. The RDX is recrystallized from cyclohexanone. Further details of the manufacture of RDX are discussed in Lindner (1980).

In the United Kingdom, the Woolwich process is used for making RDX (Lindner 1980). This process is based on nitrolysis of hexamine by nitric acid. The yield of RDX is 70 to 75 percent with only a trace of HMX. A continuous process based on the Woolwich process for medium scale production of RDX has been developed and is described in Lindner (1980).

HMX is manufactured by a modification of the Bachmann process using the same starting materials and similar equipment as in RDX production (Lindner 1980). The reaction temperature is lower (44 ± 1°C as compared to 68°C for RDX), and the raw materials are mixed in a two-step process.

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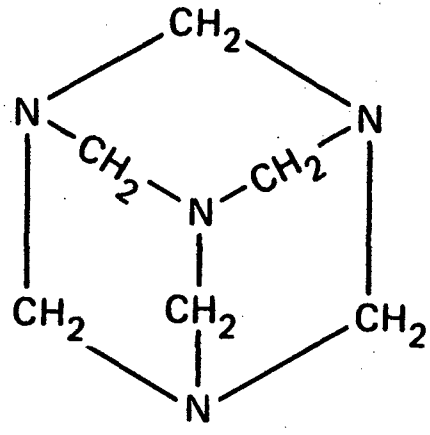
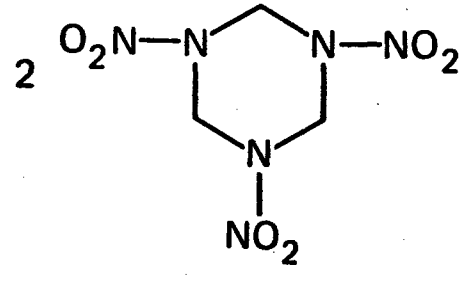


HEXAMINE

$$+ 4\ HNO_3 + 2\ NH_4NO_3 + 6(CH_3CO)_2O \xrightarrow{\Delta 68^\circ C}$$



RDX

$$+ 12\ CH_3COOH$$

Figure 3.3. Synthesis of RDX. From Fedoroff and Sheffield 1966.

The yield of HMX is 55 to 60 percent of theoretical. Further details of the manufacture of HMX are described by Lindner.

Both RDX and HMX are never handled dry because of the potential explosion hazard. They are either in a slurry stage or in solution. HMX exists in four different crystalline states of which the β form is the most stable and the least sensitive. RDX and HMX are never used alone, but are used as components with TNT and/or wax and other additives (Patterson et al. 1976).

3.3 CONCLUSIONS

Because the nature and amount of waste products are determined by the explosive manufacturing processes, the processes chosen become very pertinent to the health and environmental effects of munition production. The current method for manufacturing TNT in the United States includes the sellite purification of crude TNT which is necessary to minimize exudation. However, the sellite treatment gives rise to a waste sellite solution known as red water. Thus, alternatives to or improvement of the sellite process is important to eliminate or minimize the red water problem. The use of additives to crude TNT to minimize exudation seems to be an attractive idea.

Superior results have been claimed for the British Royal Ordnance Factory (ROF) process. During the purification of crude TNT in the ROF process, the TNT is contacted in the solid state with sellite rather than in the liquid state as used in the Radford process. This is reported to result in a purer TNT and requires only two-thirds as much sodium sulfite. Adoption of this purification procedure in the Radford process should be explored.

The current methods of manufacturing RDX and HMX appear to be satisfactory, although the potential for a significant amount of waste products does exist (see Section 8 for more information). The yields of RDX and HMX are 80-84 percent and 55-60 percent, respectively.

4. ARMY AMMUNITION PLANTS IN THE UNITED STATES: STATISTICS ON PRODUCTION RATES OF TNT, RDX, HMX, AND THE ASSOCIATED RAW MATERIALS

4.1 ARMY AMMUNITION PLANTS (AAPs) IN THE UNITED STATES

It is national policy that all munition plants be government-owned. Most of these plants are operated by contractors, typically a division of a major U.S. chemical company (GOCO, government owned and contractor operated). A few plants, mostly Navy, are operated directly by the government (GOGO, government owned and government operated). The map in Figure 4.1 shows the distribution of major AAPs and Navy Ammunition Facilities in the United States (Patterson et al. 1976).

Virtually all of the high explosives and most of the propellants for all three armed services are manufactured in the AAPs (Gilbert 1980). They are loaded in other facilities - at the same AAPs, in AAPs which do not themselves manufacture chemicals, and in a series of Naval Ammunition Facilities. The Navy facilities primarily load explosives obtained from the other AAPs. The Air Force does not manufacture explosives and propellants. They do oversee the manufacture of large solid-fueled rocket motors such as Minuteman, and there are a few GOCO facilities for this purpose. Most of the wastewater effluents result from the AAPs with a significant contribution from the Navy facilities.

As of 1976, the Army had 17 operational ammunition plants and one under construction in Mississippi. Of these, seven are engaged in manufacture, eight in load, assemble, and pack operations (LAP), and two in both manufacture and LAP operations.

4.2 PRODUCTION RATES OF TNT, RDX, HMX, AND RAW MATERIALS USED

Figure 4.2 indicates graphically the production of TNT, RDX, and HMX in the United States (Patterson et al. 1976). As shown, the amount of TNT produced as well as the capacity for production of TNT for 1969-1971 was much greater than that for both RDX and HMX.

Assuming a production rate of 50 million pounds of TNT per month by the continuous method, the raw materials used in the process are given below (calculation based on data in Gilbert 1980).

Toluene	24.2×10^6 lb	
40% Oleum	126.0×10^6 lb	
99% Nitric acid	39.4×10^6 lb	
60% Nitric acid	27.1×10^6 lb	(16.4×10^6 lb of 99% nitric acid)
Sodium sulfite . . . . (sellite purification)	5×10^6 lb	
Total	211×10^6 lb	

For every 50 million pounds of crude TNT produced, 211 million pounds of raw materials (excluding water in 60 percent nitric acid) are used, and 161 million pounds of potential wastes are created. Most of this amount is unconsumed sulfuric acid and the remainder of the toluene

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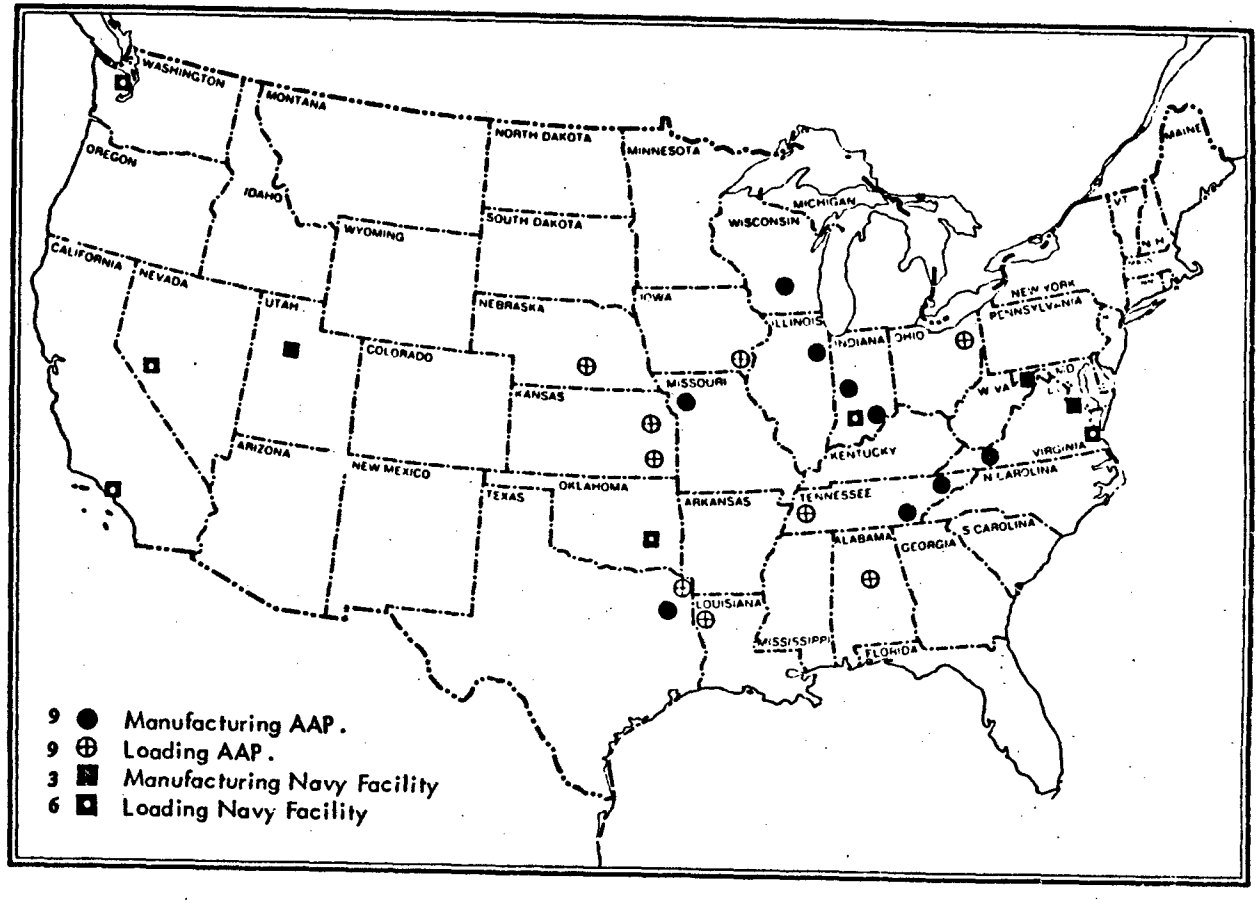

Figure 4.1. Major explosives and propellant facilities in the United States. From Patterson et al. 1976.

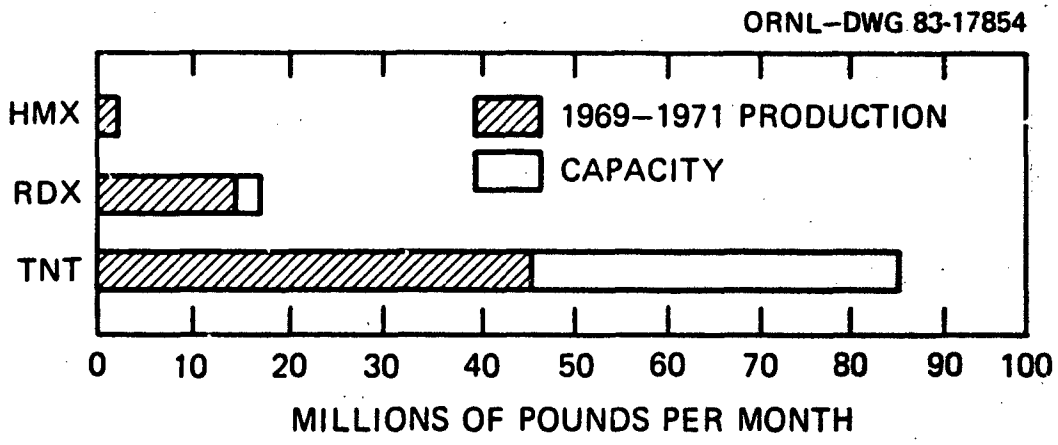

Figure 4.2. Production of TNT, RDX, and HMX in the United States. Data from Patterson et al. 1976.

and unconsumed nitric acid, as well as the spent sellite solution (red water), which contains the impurities extractable from the crude TNT (see Table 3.3). Most of this 161 million pounds is, however, either treated or recycled (see Section 9).

Assuming a production rate of 15 million pounds of RDX per month and a yield of 80 percent based on hexamine in the Bachman process, one can calculate the amount of raw materials used in its manufacture according to the following reaction:

$$C_6H_{12}N_4 + 4\ HNO_3 + 2\ NH_4NO_3 + 6\ (CH_3CO)_2O \rightarrow 2(CH_2NNO_2)_3 + 12\ CH_3COOH$$

The raw materials used are:

Hexamine	5.92×10^6 lb
Acetic acid	9.76×10^6 lb
Nitric acid	11.83×10^6 lb
Ammonium nitrate	8.88×10^6 lb
Acetic anhydride	30.77×10^6 lb
Total	67.16×10^6 lb

The total amount of by-products generated thus equals 52.17×10^6 lb ($67.17 \times 10^6 - 15 \times 10^6$). The major by-product is acetic acid which is mostly recovered, converted into acetic anhydride, and reused. Some of the by-products potentially present are listed in Section 3.2 and in Table 8.12.

The manufacture of HMX uses the same raw materials as in RDX production, and the by-products are similar.

The production of nitration acids for 1967-1971 for use in AAPs is shown in Figure 4.3. With increased recovery and reuse, the rate of production of sulfuric acid in munition plants for explosive manufacture is expected to decline. The other acid involved in the production of munitions but not identified in Figure 4.3 is acetic acid, which is used in the manufacture of RDX and HMX. It is recovered and purified for reuse, but not manufactured at the AAP (Patterson et al. 1976).

4.3 CONCLUSIONS

For 1969-1971 production of TNT was approximately 50 percent of capacity or 45 million pounds per month. RDX was produced in smaller amounts, approximately 15 million pounds per month, and HMX in still smaller quantities, less than 5 million pounds per month. A significant amount of raw materials is required, especially for TNT manufacture. The nitration acids used to produce TNT are manufactured at the AAPs, and the volume required is quite significant, sulfuric acid production being about four times the tonnage of TNT.

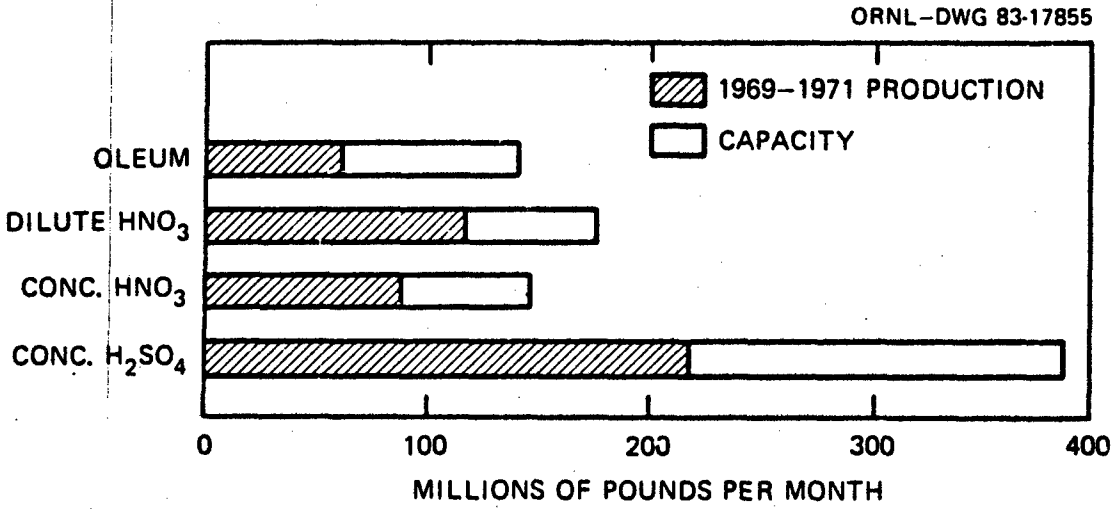

Figure 4.3. Production of nitration acids. From Patterson et al. 1976.

5. CHARACTERIZATION OF WASTE PRODUCTS FROM ARMY AMMUNITION PLANTS

5.1 INTRODUCTION

Characterization of waste products from the manufacture of TNT, RDX, and HMX is described.

5.2 WASTE PRODUCTS FROM THE MANUFACTURE AND PROCESSING OF TNT

The by-products formed during the manufacture and purification of TNT by the sellite process have been identified (Section 3.1.3). As discussed by Gilbert (1980), the maximum nominal concentration of impurities present in crude TNT is approximately 7.53 percent. This is reduced to 3.24 percent by the sellite purification step. However, of this amount, about 1 percent is not originally present in the crude TNT, but is created during sellite treatment. The amount of meta TNT isomers is reduced from 4.8 percent in crude TNT to 0.6 percent in the finished product. The control of pH during the sellite purification step is very important. At a pH of about 7.5, there is considerable loss of yield due to the formation of the water-soluble complex of 2,4,6-TNT with sodium sulfite; in addition, at pH levels above 8.0, the formation of two by-products, hexanitrobibenzyl and 3-methyl-2',4,4',6,6'-pentanitrodiphenylmethane, increases sharply.

Several gaseous pollutants arise from the manufacture of TNT with two principal species being nitrogen oxides (NO_x) and tetranitromethane (see Section 8 for more information). However, the pollutants of greatest concern in TNT manufacture and LAP (load, assemble, and pack) operations are aqueous (Lindner 1980, Pearson et al. 1979). These waste products are known as red water and pink water.

5.2.1 Red Water

During the mixed acid nitration of toluene to make TNT by the Radford continuous process, the approximate maximum concentration of by-products formed is 7.5 percent (Gilbert 1980). This is reduced to 2.4 percent by treatment with 16 percent aqueous sellite (sodium sulfite-sodium carbonate solution) on a 1:1 weight basis with crude TNT (Gilbert 1980, Hall and Lawrence 1976). The brick-red extract is known as red water or spent sellite. Red water, as obtained from the Joliet AAP in May 1975 by Hall and Lawrence (1976), was found to consist of a dark, red-brown, aqueous solution (pH 8.6) and a tan organic solid. The nitroaromatics in red water are degraded (possibly by the formation of Meisenheimer complexes followed by the loss of NO_2 groups), unless stabilized by acidification, which stops the degradation (Gilbert 1977) and also decomposes any Meisenheimer compounds (Figure 5.1) to the parent nitroaromatics. The composition of red water varies from plant to plant and also from batch to batch (Gilbert 1977).

The fractionation of red water is shown in Figure 5.2 (Hall and Lawrence 1976). TNT is the largest single nonpolar component. The total amount of TNT recoverable from red water is equal to 0.6 percent

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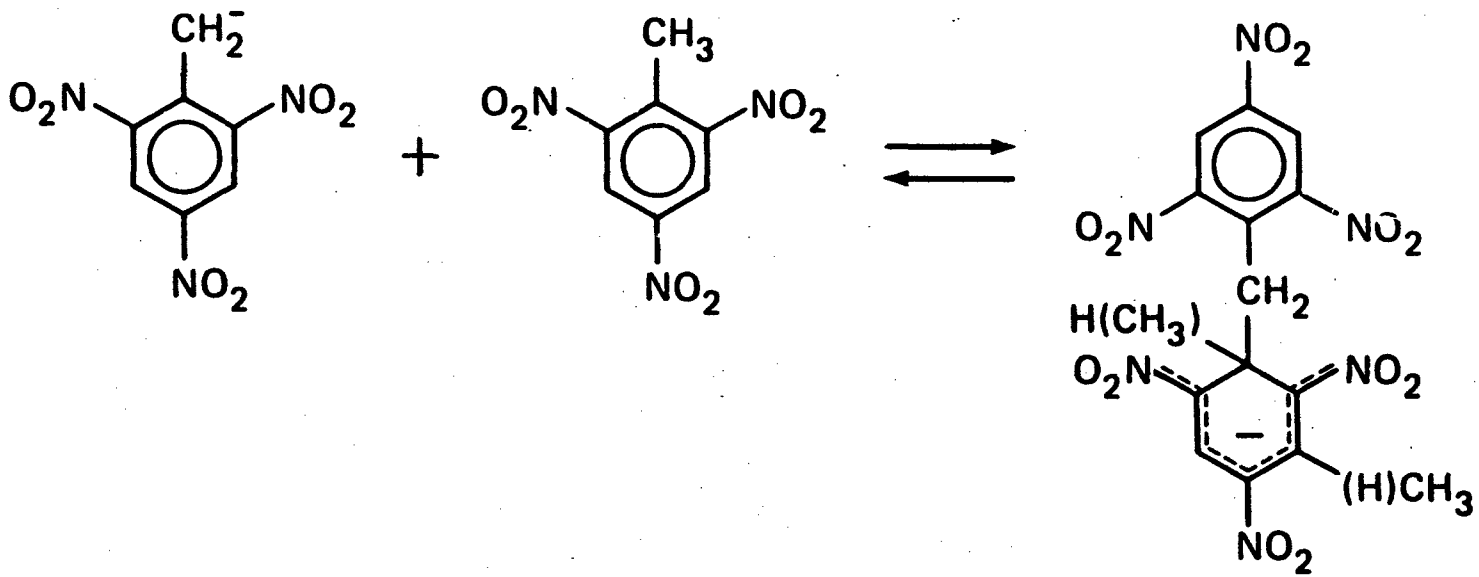


Figure 5.1. Jackson-Meisenheimer Complex, also known as Janovsky Complex. From Gilbert 1980, citing data of Bernaseoni 1971.

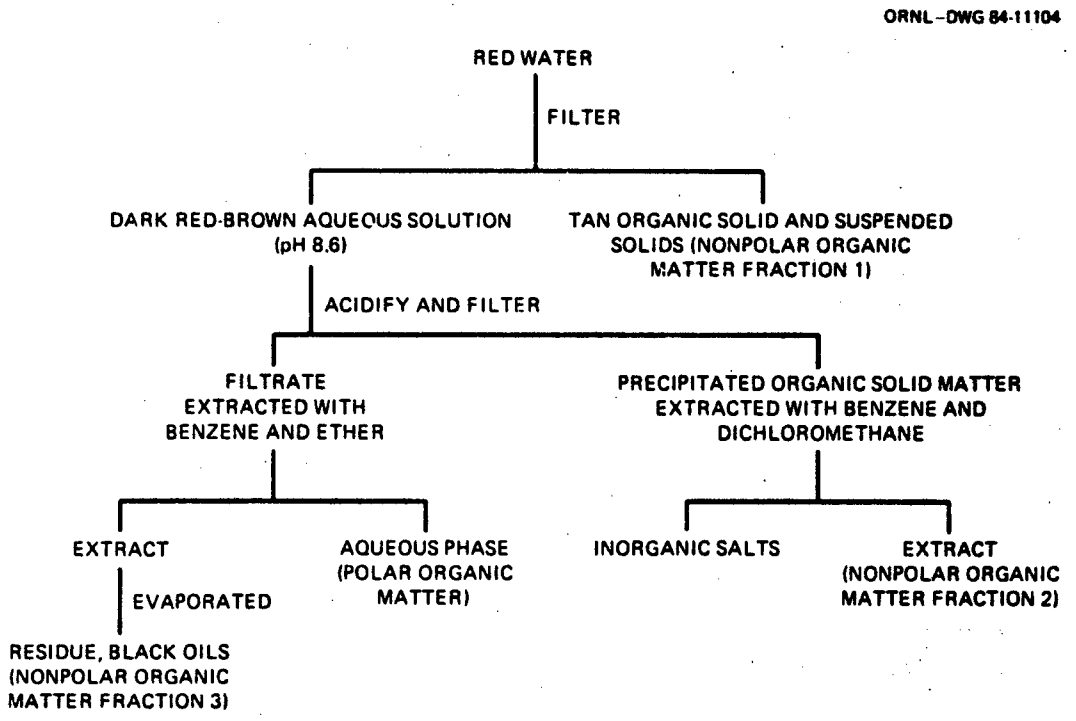


Figure 5.2. Fractionation of red water. (Fractions 1, 2, and 3 have been analyzed by thin-layer chromatography.) From Hall and Lawrence 1976.

by weight, with the majority present as suspended solids (organic fraction 1 in Figure 5.2). TNT is unstable in acidified red water; the loss after one week is 10 percent at pH 4.0 and 13 percent at pH 1.9. The nonpolar organic fractions 1, 2, and 3 (Figure 5.2) constitute about 0.62, 0.22, and 0.25 weight percent of red water, respectively.

Analyses of the Joliet AAP red water by Hall and Lawrence showed that the polar organic content of red water is 13.1 weight percent. A mixture of dinitrotoluene sulfonic acids constitutes about one-third of this fraction. These sulfonic acids are composed mainly of 2,4-dinitrotoluene-5-sulfonic (2.7 weight percent of red water) and 2,4-dinitrotoluene-3-sulfonic (2.0 weight percent of red water) acids, originating from the reaction of sodium sulfite with 2,4,5- and 2,3,4-trinitrotoluene, respectively. The remaining two-thirds of the polar fraction consists of a complex mixture of red, water-soluble matter resulting from the degradation of 2,4,6-TNT by sellite.

5.2.2 Pink Water

Pink waters are aqueous effluents that result from TNT contact with plant clean-up and scrubber water during manufacturing and LAP operations and as a condensate from red water evaporative concentration and incineration (Patterson et al. 1976). They are initially almost colorless but assume the pink characteristic color when exposed to sunlight at pH 7 and above. The color is due to the photodegradation products of TNT and is visible even at initial TNT concentration as low as 0.5 ppm. The pink constituents are a group of ill-defined water-soluble (and organic-insoluble) anions, some of which display free-radical characteristics, and they appear to constitute the major proportion of the products of TNT photolysis. Another group of photolytic TNT degradation products is extractable from water by organic solvents (Dacre and Rosenblatt 1974); most of the components of this fraction have been identified (Table 5.1). In addition to TNT, other constituents initially present in the wastewaters remain after the pink color becomes manifest with the concomitant reduction in TNT. These constituents vary with the source as shown in Table 5.2. The most important, from a toxicological point of view, appear to be the dinitrotoluene (DNT) isomers, especially 2,4-DNT and 2,6-DNT.

TABLE 5.1. PHOTODEGRADATION PRODUCTS IDENTIFIED IN ORGANIC SOLVENT EXTRACTS OF PINK WATER

Photolysis Products	In Pink Water[a]	In Photolyzed TNT/RDX Mixtures[b]
1,3,5-Trinitrobenzene	x	x
2,4,6-Trinitrobenzaldehyde	x	x
2,4,6-Trinitrobenzonitrile	x	x
4,6-Dinitroanthranil	x	x
4,4'-Dimethyl-3,3',5,5'-tetranitro-azoxybenzene	x	
3-Unidentified azoxy compounds	x	
2,4,6-Trinitrobenzyl alcohol		x
Unchanged constituents, e.g, DNT-isomers	x	
3,37,5,5'-Tetraniroazoxybenzene-2,2'-dicarboxylic acid		x
3,3',5,5'-Tetranitroazobenzene-2,2'-dicarboxylic acid		x
3,3',5,5'-Tetranitroazoxybenzene-2-carboxylic acid		x

a. Data from Dacre and Rosenblatt 1974.

b. Data from Spanggord et al. 1978.

TABLE 5.2. CONSTITUENTS OF PINK WATER (OTHER THAN THOSE DERIVED FROM α-TNT) FROM VARIOUS SOURCES[a]

Source	Constituents[b]
Manufacture	
Nitrator fume scrubbers	DNTs (MNTs), (all TNT isomers), dinitro-m-cresols[c])
Red water concentrator distillate	DNTs
Mahon fog filter effluent[d]	DNTs, (MNTs), (all TNT isomers), (dinitro-m-cresols[c])
Finishing building air scrubbers and washdown[e]	(DNTs)
RDX/TNT incorporation[f]	RDX, HMX, and products associated with their manufacture
Load, assemble, and pack plants	RDX, HMX

a. Data from Dacre and Rosenblatt 1974.

b. Parentheses mean constituents are believed present but not positively identified.

c. Nitrocresols could arise by displacement of a nitro group on any of the isomers of alpha-TNT.

d. Volunteer AAP only.

e. Alpha-TNT from this source is rather pure.

f. Holston AAP.

The colored photodegraded mixture (water soluble and organic insoluble fraction of pink water) has a lower acute mammalian toxicity than does a solution of α-TNT. The mouse oral LD_{50} value of 100 percent degraded TNT is about four times that of the original material (Rosenblatt 1980). This seems to be the rationale for directing a major effort towards evaluating the toxicity due to the organic soluble fraction of the pink water (Pearson et al. 1979, Spanggord et al. 1978). Because the composition of pink water varies from plant to plant, an attempt has been made to do the toxicological studies with a synthetic condensate wastewater mixture. Condensate water is formed when red water is concentrated by evaporation. As many as thirty components have been identified and quantitated in samples of condensate discharge at Volunteer AAP (Table 5.3). Possible sources of these components and their formation in the condensate water from precursors have been discussed by Spanggord et al. (1978). The components have been separated by gas chromatography and identified by mass spectrometry and PMR with authentic compounds.

TABLE 5.3. THE 90TH PERCENTILE CONCENTRATIONS AND RELATIVE CONCENTRATIONS DETERMINED FOR CONDENSATE COMPONENTS[a]

Condensate Component	90th Percentile Concentration (mg/L)	Relative Concentration (%)
Toluene	0.200	0.590
2-Nitrotoluene (NT)	0.030	0.089
4-Nitrotoluene	0.100	0.295
3-Nitrobenzonitrile[b]	0.013	0.035
4-Nitrobenzonitrile[b]	0.009	0.027
2-Amino-4-NT	0.033	0.097
2-Amino-6-NT[b]	0.010	0.030
3-Amino-4-NT[b]	0.027	0.080
3-Methyl-2-nitrophenol	0.012	0.035
5-Methyl-2-nitrophenol	0.032	0.094
1,3-Dinitrobenzene (DNB)	4.000	11.803
2,3-Dinitrotoluene (DNT)	0.400	1.180
2,4-DNT	14.700	43.377
2,5-DNT	0.400	1.180
2,6-DNT	7.300	21.541
3,4-DNT	0.500	1.475
3,5-DNT	0.520	1.534
3,5-Dinitroaniline[b]	0.058	0.171
1,5-Dimethyl-2,4-DNB (DNX)	0.390	1.151
2-Amino-3,6-DNT	0.030	0.089
2-Amino-4,6-DNT	0.020	0.059
3-Amino-2,4-DNT	1.500	4.426
3-Amino-2,6-DNT	1.200	3.541
4-Amino-2,6-DNT	0.600	1.770
4-Amino-3,5-DNT	0.200	0.590
5-Amino-2,4-DNT	0.700	2.066
2,4-Dinitro-5-methylphenol[b]	0.085	0.251
1,3,5-Trinitrobenzene (TNB)[b]	0.153	0.451
2,3,6-Trinitrotoluene (TNT)[b]	0.268	0.791
2,4,6-TNT	0.400	1.180

a. Data from Spanggord et al. 1978.

b. Compounds were not present in 10% of the samples. Value given represents the mean of the nonzero values.

5.3 WASTE PRODUCTS FROM THE MANUFACTURE AND PROCESSING OF RDX AND HMX

Significant quantities of wastes are created during the manufacture and LAP operations involving RDX and HMX. Both gaseous and aqueous effluents are generated, and these contain both organic (including the munitions themselves and the two principal by-products, TAX and SEX) and inorganic species. Section 8 of this report presents a more detailed characterization of individual compounds and elements.

Patterson et al. (1976) state that the major wastewater sources from RDX and HMX manufacture result from dewatering of the explosives in special vacuum carts (nutsches), decanting water from hot composition explosive blending, and floor and equipment cleanup wash water. Explosives-contaminated wastewater also results from dust control by scrubbers at explosives packaging buildings. According to Patterson et al. (1976), limited information is available on wastewater characteristics associated with the handling and loading of RDX- and HMX-based explosives at LAP facilities.

5.4 CONCLUSIONS

Red water has not been fully characterized. The major organic components identified are 2,4-dinitrotoluene-3-sulfonate and 2,4-dinitrotoluene-5-sulfonate (Gilbert 1977). The evaporator condensate of red water has, however, been examined rather thoroughly.

The fraction of pink water which is water soluble but not extractable by organic solvents and which is responsible for the pink color (due to the photodegradation of TNT) has not been examined for toxicity, presumably because 100 percent photodegraded TNT is one-fourth as toxic as the parent material. Attempts should be made, however, to chemically characterize this fraction because of possible synergistic effects of the components of pink water. The fraction of the pink water extractable with organic solvents has been well characterized (Spanggord et al. 1978).

6. METABOLISM OF TNT, RDX, HMX, AND WASTE CONSTITUENTS

6.1 METABOLISM OF TNT

TNT exposures can occur by inhalation of the dust, ingestion, or skin absorption. Although the mechanism of metabolism is unknown, it is generally accepted that TNT is rapidly detoxified in the liver and excreted primarily by the kidneys (Jaffe et al. 1973; Dacre and Rosenblatt 1974). Only small amounts of the compound or its metabolites are distributed to the organs (Table 6.1). Following oral administration to the rat, El-Hawari et al. (1978) found that males and females, respectively, excreted 59.9 percent and 42.2 percent in the urine and 10.8 percent and 2.2 percent in the feces, with 21.0 percent and 35.3 percent remaining in the gastrointestinal tract at the end of 24 hours. Absorption and elimination were faster after intratracheal instillation and slower after dermal application.

TABLE 6.1. DISTRIBUTION AND EXCRETION OF RADIOACTIVITY IN RATS RECEIVING A SINGLE ORAL DOSE OF ^{14}C-TNT[a]

Source	Percentage of Administered Dose	
	30 minutes	24 hours
Gastrointestinal tract plus contents	-	20.7 ± 2.7
Feces	-	5.5 ± 1.1
Whole blood	0.2 ± 0.0	0.6 ± 0.1
Expired air	-	0.1 ± 0.0
Urine	0.2 ± 0.1	53.5 ± 3.1
Liver	0.3 ± 0.1	0.6 ± 0.0
Kidneys	0.1 ± 0.0	0.2 ± 0.0
Brain	0.1 ± 0.0	<0.1
Lungs	-	<0.1
Skeletal muscle	-	1.0 ± 0.1
Recovery		82.1 ± 3.0

a. Data from Zakhari et al. 1978, citing Lee et al. 1975.

The type and quantity of metabolites identified in the urine of TNT workers and of experimental animals receiving TNT by several routes indicate that TNT is metabolized primarily by reduction of the nitro groups and, to a lesser extent, by oxidation of the methyl group and ring hydroxylation (Dilley et al. 1978a; Rosenblatt 1980). The presence of unidentified glucuronide conjugates indicates the formation of either trinitrobenzyl alcohol or aminodinitrobenzyl alcohol.

Metabolites identified from several sources are listed in Table 6.2. The main metabolite identified in biochemical studies is 4-amino-2,6-dinitrotoluene. Metabolites found in human urine are the same as those

TABLE 6.2. METABOLITES OF TNT

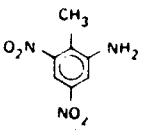
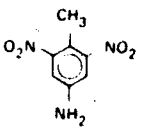
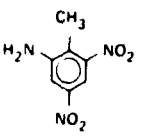
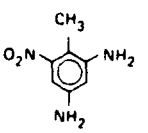
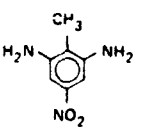
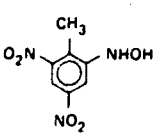
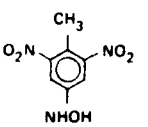
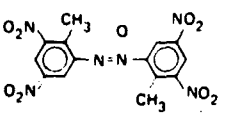
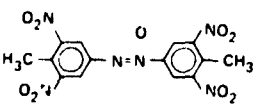
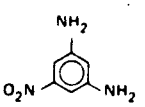
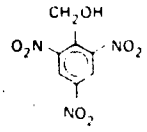
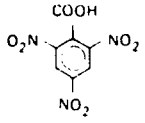
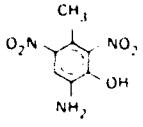
METABOLITES	SOURCES					
	HUMAN	RABBIT	RAT	REACTION WITH XANTHINE/ XANTHINE OXIDASE	PIG LIVER EXTRACT	BACTERIA
1. Monoaminodinitrotoluenes						
2 Amino 4,6 dinitrotoluene 						X[a]
4 Amino 2,6 dinitrotoluene 	X[b,c]	X[d]	X[b,e]		X[f]	X[a,g]
6 Amino 2,4 dinitrotoluene 	X[b]	X[d]	X[e]			
2. Diaminomononitrotoluenes						
2 4 Diamino 6 nitrotoluene 	X[c]		X[b,e]			X[g]
2,6 Diamino 4 nitrotoluene 			X[e]			
Unspecified isomer						X[a]
3. Hydroxylaminodinitrotolueres						
2 Hydroxylamino 4,6 dinitrotoluene 		X[h]	X[h]			
4 Hydroxylamino 2,6 dinitrotcluene 	X[b]	X[d]		X[f]		X[a]
4 Tetranitrozoxytoluenes'						
2,2',4,4' Tetranitro 6,6' azoxytoluene 						X[a]
2,2',6,6' Tetranitro 4,4' azoxytoluene 		X[d]				X[a]
5. 3,5 Diaminonitrobenzene 			X[b]			

TABLE 6.2. METABOLITES OF TNT (continued)

METABOLITES	SOURCES					
	HUMAN	RABBIT	RAT	REACTION WITH XANTHINE/ XANTHINE OXIDASE	PIC LIVER EXTRACT	BACTERIA
6 2,4,6 Trinitrobenzyl alcohol 		X[h]	X[h]			
7 2,4,6 Trinitrobenzoic acid 		X[h]	X[h]			
8 Amino nitrocresols 	X[b]		X[b]			
9 Glucuronides		X[d]	X[e]			
10 Succinic acid + amino acids						X[g]
11 Nitrite, nitrate ions						X[f]

a. Won et al. 1974
b. Lemberg and Callaghan 1944.
c. Horecker and Snyder 1944, as reviewed in Zakhari et al. 1978.
d. Channon et al. 1944.
e. El hawari et al. 1978.
f. Bueding and Jolliffe 1946.
g. Fowler 1965, as reviewed in Dacre and Rosenblatt 1974.
h. Cited in Rosenblatt 1980, may be formed in one or both species.
i. Formed in standing urine and bacterial cultures, probably from the condensation of hydroxylaminodinitrotoluenes.

found in rabbit urine, namely 4-hydroxylamino-2,6-dinitrotoluene, 4-amino-2,6-dinitrotoluene, and 6-amino-2,4-dinitrotoluene. Bueding and Jolliffe (1946) achieved nearly quantitative reduction of TNT to 4-hydroxylamino-2,6-dinitrotoluene and 4-amino-2,6-dinitrotoluene in vitro by treatment with xanthine-xanthine oxidase in the first case and pig liver extract in the second. Nearly half of the administered dose is excreted as glucuronides as illustrated by the rabbit (Channon et al. 1944).

Proposed routes of metabolism are outlined in Figure 6.1. Reduction of the 4-nitro group is illustrated in the first step, but reduction also takes place at the 6-position and, to a lesser extent, at the 2-position as indicated by the metabolites in Table 6.2. Two of the metabolites, 2,4,6-trinitrobenzoic acid and 2,4,6-trinitrobenzyl alcohol, have been only tentatively identified in urine. Their presence is, however, supported by the fact that 2,4,6-trinitrobenzyl alcohol produces a red pigment in the urine of experimental animals administered TNT, a phenomenon not shown by other nitro compounds (Channon et al. 1944). The presence of glucuronide conjugates in the urine also supports the formation of an oxidation product such as trinitrobenzyl alcohol. The formation of 3,5-diaminonitrobenzene most likely involves oxidation of trinitrobenzyl alcohol to trinitrobenzoic acid, followed by decarboxylation to trinitrobenzene and reduction to diaminonitrobenzene (Jaffe et al. 1973).

6.2 METABOLISM OF RDX

RDX (hexahydro-1,3,5-trinitro-1,3,5-triazine) is slowly absorbed from the stomach after ingestion and also apparently from the lungs after inhalation; there is little evidence of skin absorption. It is extensively metabolized and does not accumulate in any tissue.

Twenty-four hours after the oral administration of 50 mg/kg of ^{14}C-RDX to rats, 37-39 percent of the total radioactivity was still present in the gut, but less than 3 percent was present after 48 hours (Schneider et al. 1976a, 1976b). Four days after administration, less than 10 percent of the original dose remained in all tissues of the rat. Three percent had been excreted unchanged, primarily in the urine. The remainder was metabolized, and the metabolites were excreted either in the urine (31 percent) or feces (3 percent) or exhaled as $^{14}CO_2$ (43 percent).

Irrespective of dosage or route of administration, the concentration of RDX in the rat following single doses was greatest in the kidney, was most variable in the liver, and, based on tissue/plasma ratios, accumulated to some extent in all tissues examined (Schneider et al. 1977). Tissue distributions at 2 to 24 hours after oral dosing are given in Table 6.3. Following single oral doses to miniature swine and subchronic oral administration to rats, a similar extensive pattern of RDX metabolism was found; but, in contrast to the results from single oral doses to rats, no single tissue had higher RDX concentrations than other tissues (Schneider et al. 1977, 1978).

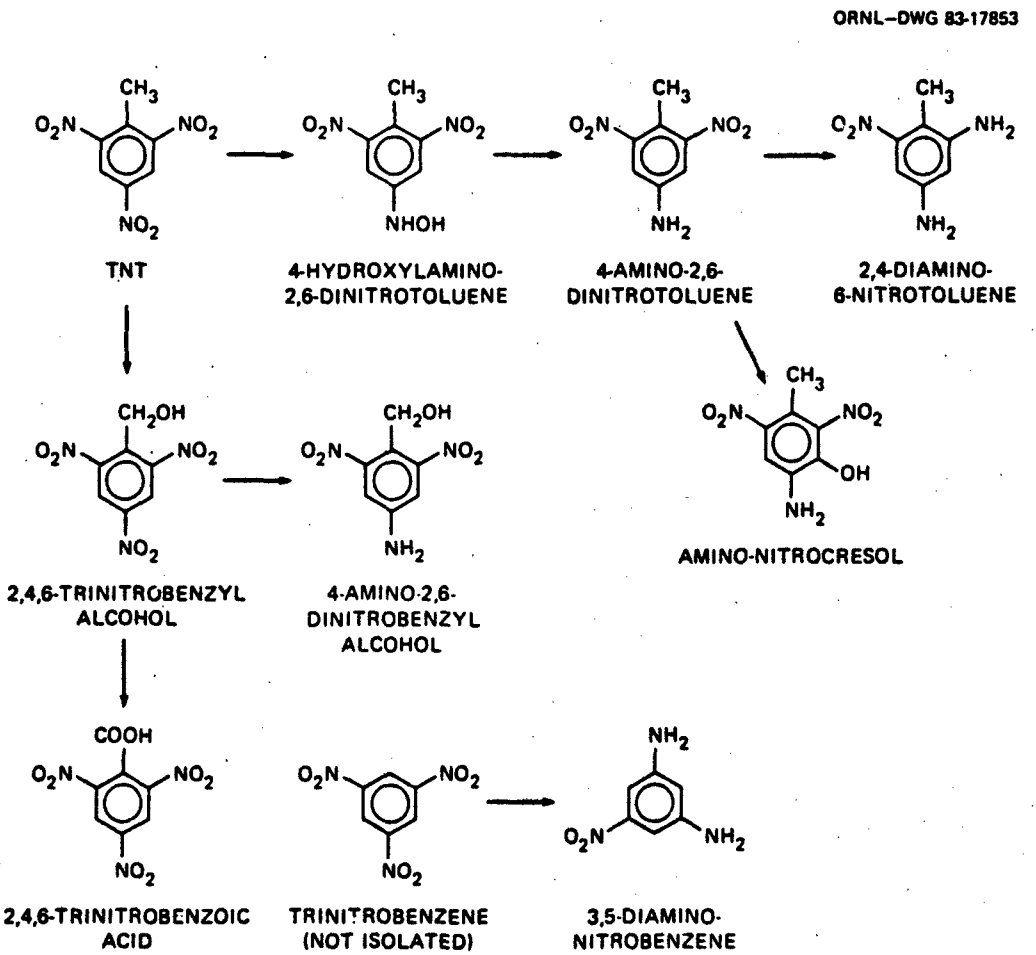


Figure 6.1. Proposed routes of metabolism of TNT based on urinary metabolites.

TABLE 6.3. PLASMA, URINE, AND TISSUE RDX CONCENTRATIONS AT VARIOUS TIMES AFTER DOSING RATS WITH 100 MG/KG BY GAVAGE[a]

Time (hr)	Plasma (μg/g)[b]	Urine (μg/mL)	Brain (μg/g)	Heart (μg/g)	Liver (μg/g)	Kidney (μg/g)
2	1.50 ± 0.26	2.45 ± 0.30	10.36 ± 1.24	7.97 ± 1.11	4.34 ± 0.90	12.86 ± 1.40
4	2.09 ± 0.09	5.46 ± 0.96	7.71 ± 0.98	6.49 ± 0.96	2.16 ± 0.56	12.30 ± 1.82
6	1.78 ± 0.15	5.02 ± 0.81	7.51 ± 0.57	7.13 ± 0.71	0.51 ± 0.34	13.58 ± 1.37
8	2.36 ± 0.22	7.31 ± 0.85	5.57 ± 0.67	3.82 ± 0.52	0.15 ± 0.15	10.90 ± 0.86
12	2.26 ± 0.16	5.49 ± 1.03	11.28 ± 1.60	11.08 ± 1.82	8.51 ± 2.24	22.02 ± 2.06
18	2.03 ± 0.10	5.58 ± 0.28	6.30 ± 0.20	5.58 ± 2.24	0.48 ± 0.20	12.12 ± 0.83
24	3.04 ± 0.48	6.87 ± 0.84	8.91 ± 1.07	7.89 ± 0.83	2.56 ± 1.15	16.85 ± 0.80

a. Data from Schneider et al. 1977.

b. Conversion factor (micrograms per milliliter to micrograms per gram) = 0.9737.

In the rat, plasma concentrations reached a dose-dependent plateau within several hours, were maintained at these levels for 24 hours, and then declined over the next two days (Schneider et al. 1977). In miniature swine, absorption appeared to be slower; plasma concentrations peaked between 12 and 24 hours after administration, at which time the animals were sacrificed.

Based on the following observations it is generally concluded that the reactions involved in RDX metabolism are catalyzed by microsomal enzyme systems and occur primarily in the liver. After oral administration, greater amounts of radioactivity were found in the liver than were accounted for by its RDX content, indicating the presence of RDX metabolites (Schneider et al. 1977). A single oral dose to rats caused extensive long-lasting proliferation of the smooth endoplasmic reticulum in the liver, indicating the possible induction of the mixed function oxidase (MFO) system (French et al. 1976). Phenobarbital, an MFO inducer, increased the rate of RDX metabolism while pyrazole, an MFO inhibitor, decreased the RDX metabolic rate (Bradley 1977). In rat liver microsomal enzyme assays, RDX acted to a limited extent as a microsomal enzyme inducer as evidenced by the stimulation of the metabolism of o-nitroanisole (O-demethylation) (Dilley et al. 1978a). It showed no stimulatory activity in the metabolism of aminopyrene (N-demethylation) or aniline (aromatic hydroxylation).

Metabolism of RDX produces several one-carbon fragments: CO_2 (Schneider et al. 1977), bicarbonate ion, and formic acid (Schneider et al. 1978, citing unpublished observations of Andersen and Bradley). No larger intermediates have been identified.

6.3 METABOLISM OF HMX

No information on the metabolism of HMX (octahydro-1,3,5,7-tetranitro-1,3,5,7-tetrazine) was available from the published literature. Its primary toxic effect - cardiovascular depression - is characteristic of nitrite toxicity indicating cleavage of the NO_2 groups. However, no methemoglobin is formed (McNamara et al. 1974).

6.4 METABOLISM OF WASTE CONSTITUENTS

6.4.1 Dinitrotoluenes

Dinitrotoluene (DNT) compounds are well absorbed after oral dosing of mammals, extensively metabolized in the liver, and rapidly excreted in the urine and, to a lesser extent, in the bile (Ellis et al. 1980). There is also evidence that cecal flora metabolize DNT via an ordered sequence of reductive steps, and the cecum may represent a major site of reductive metabolism of DNT (Dent et al. 1982). Radioactivity of ^{14}C-labeled compounds is widely distributed in the carcass, but concentrates only in the liver and kidney. Very little of the parent compounds are found in the urine.

The urinary metabolites derived from 2,4-DNT, the major component of red water distillate, have been studied in detail following single-dose

and chronic feeding studies (Ellis et al. 1980, citing data of Lee et al. 1975, 1977). There are two primary reactions: reduction of one or both nitro groups to amino groups and oxidation of the methyl side chain through benzyl alcohol and benzaldehyde to benzoic acid. This results in 16 possible metabolic products (Figure 6.2). Most of the compounds undergo conjugation with glucuronide or sulfate before excretion. The major metabolites in chronic feeding studies with rats were dinitro-(V), aminonitro-(VI and VII), and diaminobenzyl(VIII) alcohols (Ellis et al. 1979).

The disposition of radioactivity in rats given oral doses of ^{14}C-ring-labeled DNT isomers (i.e., the 2,3-, 2,5-, 2,6-, 3,4-, and 3,5-isomers) was similar to that of 2,4-DNT and TNT (Ellis et al. 1980). The compounds were well absorbed 24 hours after dosing, widely distributed with higher concentrations in the liver and kidneys, and excreted primarily in the urine. For all compounds, negligible amounts of radioactivity were expired as CO_2, indicating that the aromatic ring remained intact.

6.4.2 Other By-products and Waste Constituents

No information on the metabolism of TNT isomers is available. All five meta isomers of TNT are formed during its manufacture by the Radford continuous process. Of these, 2,4,5-trinitrotoluene (γ) and 2,3,4-trinitrotoluene (β) are the most abundant and are present in the crude TNT to the extent of 2.5 percent and 1.75 percent, respectively. These isomers are mostly converted into 2,4-dinitrotoluene-5-sulfonate and 2,4-dinitrotoluene-3-sulfonate during sellite treatment (see Section 3.1.4).

No information is available on the metabolism of tetranitromethane, trinitromethane, or methyl nitrate. The induction of methemoglobin in many toxicity studies with these compounds suggests cleavage of the NO_2 group which is converted to nitrate and nitrite ions. The mechanism of oral toxicity in tetranitromethane administration may be nitrite-induced methemoglobinemia.

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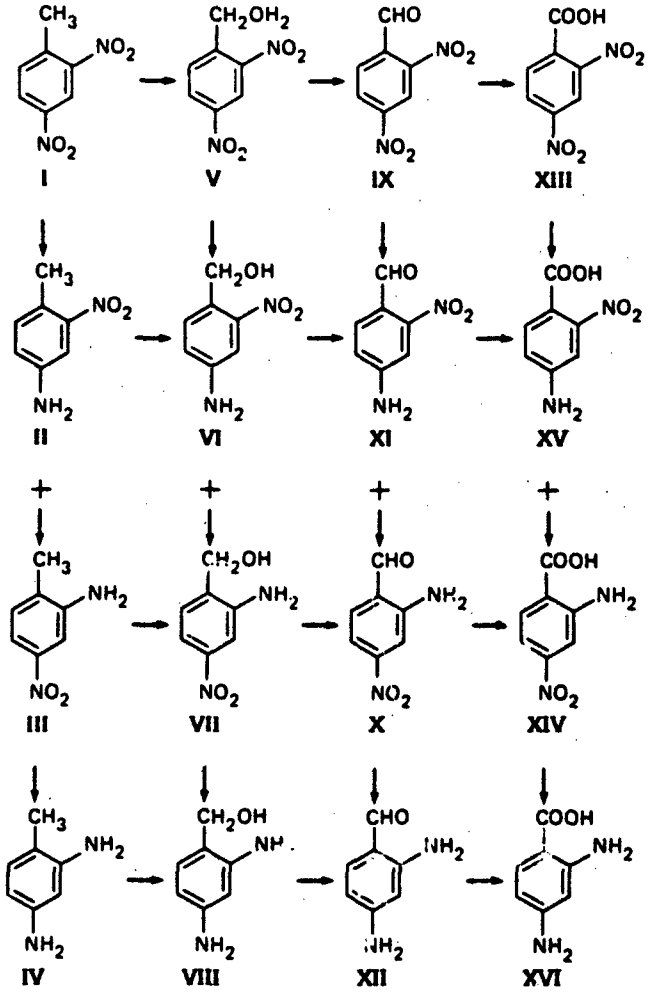

Figure 6.2. Possible early steps of metabolic pathways of 2,4-DNT in mammals. The compounds are: I = 2,4-dinitrotoluene; II = 4-amino-2-nitrotoluene; III = 2-amino-4-nitrotoluene; IV = 2,4-diaminotoluene; V = 2,4-dinitrobenzyl alcohol; VI = 4-amino-2-nitrobenzyl alcohol; VII = 2-amino-4-nitrobenzyl alcohol; VIII = 2,4-diaminobenzyl alcohol; IX = 2,4-dinitro-benzaldehyde; X = 4-amino-2-nitro-benzaldehyde; XI = 2-amino-4-nitro-benzaldehyde; XII = 2,4-diamino-benzaldehyde; XIII = 2,4-dinitrobenzoic acid; XIV = 4-amino-2-nitrobenzoic acid; XV = 2-amino-4-nitrobenzoic acid; XVI = 2,4-diaminobenzoic acid. From Ellis et al. 1980.

7. HEALTH EFFECTS IN HUMANS AND IN EXPERIMENTAL ANIMALS OF INDIVIDUAL EXPLOSIVES AND THEIR BY-PRODUCTS

7.1 TNT

TNT is of concern because of its known toxic effects to humans and because of the potential for exposure of munitions industry workers to dust and fumes during manufacturing, shell-loading, and clean-up processes. Humans may also be exposed to low levels of TNT and its isomers as well as other organic compounds in wastewaters which are discharged to the aquatic environment.

There are three possible routes through which TNT may enter the body of exposed workers. Though TNT is absorbed primarily through the skin, it can enter the body by inhalation of the fumes or dust during the nitrating or grinding processes, respectively, and by ingestion through improper hygienic procedures or the use of contaminated water supplies. Skin absorption takes place most readily through the palms of the hands, the neck, and the face (Voegtlin et al. 1921-1922).

7.1.1 Health Effects in Humans

During World War I, 24,000 cases of TNT intoxication, including 580 deaths, were reported in the United States (Zakhari et al. 1978, citing data of Hegyeli 1974). Fatalities due to TNT intoxication during World War II fell sharply, due to the enforcement of strict industrial hygiene measures. Thus, only 22 deaths were reported in the period from June 1, 1941, to September 1, 1945 (McConnell and Flinn 1946).

Among the first signs of TNT intoxication in both acute and chronic exposures are hematologic changes. In student volunteers exposed in a munitions plant to an air concentration of 0.3 mg TNT/m^3 for 8 hours/day, 5-6 days/week for an average of 33 days, the hemoglobin content and circulating red blood cell counts were reduced in 85 percent of the subjects (Stewart et al. 1945). Most of these volunteers also had a rash on their hands.

Reduction of the red blood cell count and of the hemoglobin content may be associated with chemical and physical changes in blood cells: polychromasia (variation in the hemoglobin content of the erythrocytes), poikilocytosis (presence of irregular erythrocytes of abnormal size and shape), anisocytosis (the presence of nucleated red blood cells), reticulocytosis, and eosinophilia (Voegtlin et al. 1921-1922). Changes in the white blood cell picture due to TNT exposure may consist of leukocytosis or relative lymphocytosis. Increased capillary fragility leads to nose bleeds and hemorrhages of the skin and mucosa. At high levels of exposures, methemoglobinemia with consequent cyanosis appears. Accounts from the European literature link cataracts of the eyes with chronic exposure to TNT.

Dacre and Rosenblatt (1974, citing data of Von Oettingen et al. 1944) reported observations of two human volunteers who rubbed 500 mg

(approximately 7.1 mg/kg) of finely powdered TNT into the palms of their hands. The hands were covered with rubber gloves, and the TNT was kept on the skin for 8 hours. The only effect noted was a moderately bitter taste during the course of the experiment. Traces of the metabolite 4-amino-2,6-dinitrotoluene were found in the urine during the first 23 hours following exposure.

The most serious systemic sequelae to acute TNT poisonings in humans are toxic hepatitis which ultimately leads to yellow atrophy of the liver (McConnell and Flinn 1946; Palmer et al. 1943; Evans 1941) and hypoplasia of the bone marrow which leads to aplastic anemia (McConnell and Flinn 1946; Sievers et al. 1946; Hayhoe 1953; Crawford 1954). In a study of 22 fatalities during World War II, deaths from toxic hepatitis were more common among younger age group subjects (average age 30 years) and aplastic anemia among older age group subjects (average age 45 years) (McConnell and Flinn 1946). The most important clinical finding in the fatal cases of toxic hepatitis was a high icterus (bilirubin) index.

Although TNT exerts its most severe toxic effects on the liver and the hemopoietic system, other organs and tissues are also affected. Effects observed in workers exposed to TNT have been summarized from the literature (Table 7.1). Although exposure to TNT is the main source of toxicity for workers in manufacturing plants, exposures to nitrous (NO_x) fumes during the nitration process can produce irritant effects on the lungs (Zakhari et al. 1978).

Since World War II there have been few reports of health problems or deaths related to TNT manufacturing or handling. In 1972 Goodwin reported only reversible liver damage occurring in a small percentage of workers in a shell loading plant in a period of over 20 years. Atmospheric levels of TNT were not given, but are presumed to have been below the threshold limit value of 1.5 mg/m^3 then in effect.

In order to set safe working standards, studies providing correlation between work exposures and adverse health effects must be available. Two studies relate mild biological effects to exposure levels. Exposure levels and mean hemoglobin values for more than 500 exposed employees are listed in Table 7.2. A reduction in hemoglobin levels was noted at exposures as low as 0.2 mg/m^3 (Hathaway 1977). Statistically significant rises in serum glutamic oxalacetic transaminase (SGOT) and lactic dehydrogenase (LDH) occurred at exposures of 0.8 mg/m^3 at an ammunition plant when the production rate at the fill and pack line was 126 percent production capacity. These changes persisted the following month when TNT dust levels dropped to 0.6 mg/m^3 at 100 percent production capacity (Table 7.3) (Morton et al. 1976). These enzyme changes most probably represent hepatocellular damage and some hemolysis.

Zakhari et al. (1978, citing data of Morton and Ranadive 1974) discussed epidemiological studies on 27 cases designed to determine which of the above tests (Hb, SGOT, LDH) might be used for routine monitoring

TABLE 7.1. HEALTH HAZARDS CAUSED BY ACUTE OR CHRONIC EXPOSURE TO TNT[a]

	Symptom
Liver	Acute yellow atrophy and yellow jaundice
Blood	Methemoglobinemia with the consequences of oxygen deficiency, hemolytic anemia (in glucose-6-phosphate dehydrogenase-deficient workers), aplastic anemia, and thrombocytopenia
Heart	Depression of the myocardium; various electrocardiographic changes
Blood vessels	Increased permeability to proteins
Pancreas	Exocrine dysfunction
Skin	Dermatitis, eczema
Eye	Arc-shaped cataract in the lens after forming an irregular ring
Teeth and oral cavity	Caries and stomatitis
Central nervous system	Various functional changes
Kidney	Partial retention of sodium, increased excretion of porphyrins
Biochemical changes	Increase in plasma bilirubin and possible changes in SGOT and LDH[b]
Gastrointestinal tract	Gastritis, nausea and vomiting; epigastric pain

a. Modified from Zakhari et al. 1978.

b. SGOT = serum glutamic oxalacetic transaminase;
LDH = lactic dehydrogenase.

of workers. They concluded that none of the tests was sufficiently sensitive to be used as an indicator for the detection of TNT toxicity and thus all three tests should be carried out simultaneously.

To date, there is no single and specific test to detect the early symptomless stages of TNT toxicity. A sensitive and specific test for the detection of TNT or its effects in biological fluids needs to be developed.

No carcinogenic effects have been reported from TNT intoxication among workers, and no tumors have been found in postmortem examinations of persons chronically exposed to TNT (Zakhari et al. 1978). A retrospective epidemiological study of workers involved in the production of TNT during World War II could possibly confirm this observation.

TABLE 7.2. RELATIONSHIP BETWEEN HEMOGLOBIN VALUES AND TNT EXPOSURE LEVELS[a]

Exposure Levels (mg/m^3)	Whites				Nonwhites			
	Mean Hb[b]		Relative Odds[c]		Mean Hb		Relative Odds	
	M	F	M	F	M	F	M	F
Not exposed	15.2	13.7	1.0	1.0	14.7	13.1	1.0	1.0
0.01 or less	15.0	13.6	1.1	1.4	14.7	12.9	0.8	1.5
0.02 to 0.09	14.7	13.6	1.8	7.0	14.9	13.3	0.5	0.0
0.10 to 0.19	14.7	13.5	1.9	0.0	14.3	12.9	2.1	2.5
0.20 to 0.29	14.4	13.8	2.8	0.0	13.9	—	1.2	—
0.30 to 0.39	14.0	10.6	6.9	—	13.7	—	2.5	—
0.40 to 0.49	14.8	13.6	2.9	0.0	13.8	12.8	3.5	3.8
0.50 to 0.99	14.4	13.4	6.0	8.0	13.6	13.0	7.5	—
1.00 to 1.49	13.7	—	6.2	—	—	—	—	—
1.50 and over	—	12.4	—	—	13.1	—	—	—

a. From Hathaway 1977.

b. The following numbers for Hb values were considered abnormal: males <14 g/100 mL blood; females <12 g/100 mL blood.

c. Relative odds (R.O.) is a relative number that was calculated as follows:

$$R.O. = \frac{\text{Number of control cases showing normal values x number of exposed cases showing abnormal values}}{\text{Number of control cases showing abnormal values x number of exposed cases showing normal values}}$$

7.1.2 Toxic Effects in Laboratory Animals

7.1.2.1 General Toxicity

Prior to 1975 no studies to determine the acute LD_{50} of TNT had been conducted. Most early investigators determined the lethal dose for experimental animals, but did not always clearly indicate the design of the experiment, the number of animals used, or the exact dose given via the respective routes (Zakhari et al. 1978). From these early investigations it can be concluded that (1) animal species vary considerably in their sensitivity to acute TNT poisoning, (2) there are striking differences in individual susceptibilities, and (3) most of the toxic effects observed in animals have also been observed in humans (Rosenblatt 1980).

Acute oral LD_{50} values listed in Table 7.4 indicate that TNT is only moderately toxic from single oral doses. These doses produced convulsions, respiratory inhibition, cyanosis, ataxia, and red urine (Zakhari et al. 1978, citing data of Lee et al. 1978). The red color of the

TABLE 7.3. MEAN TNT DUST LEVELS, PRODUCTION RATE AND MEAN LABORATORY TEST VALUES OF 43 EMPLOYEES EXPOSED TO TNT BY MONTH[a]

	Preemployment	12/73	1/74	2/74	3/74	4/74
Hb[b]	15.58	14.96	15.19	15.07	14.85	14.90
SD[c]	(±1.15)		(±0.72)	(±0.88)	(±0.99)	(±1.11)
No. abnormal results	2	2	2	3	3	1
SGOT[d]	34.78	35.11	32.47	39.06	58.17	54.44
SD	(±18.15)	(±16.59)	(±18.77)	(±37.06)	(±70.73)	(±50.87)
No. abnormal results	3	1	2	2	10	7
LDH[e]	64.37	52.63	51.72	50.88	105.78	102.76
SD	(±16.52)	(±12.16)	(±11.57)	(±11.02)	(±39.68)	(±40.91)
No. abnormal results	4	0	0	0	23	13
Production rate (%) capacity	N/A	75	75	75	126	100
Average TNT dust levels	N/A	0.3 mg/m^3	0.3 mg/m^3	0.3 mg/m^3	0.8 mg/m^3	0.6 mg/m^3

a. From Morton et al. 1976.

b. Hb = hemoglobin.

c. SD = standard deviation.

d. SGOT = serum glutamic oxalacetic transaminase.

e. LDH = lactate dehydrogenase.

TABLE 7.4. ACUTE ORAL LD_{50} VALUES FOR TNT

Species	LD_{50} (mg/kg)	Reference
Mouse (male)	1014	Zakhari et al. 1978, citing data of Lee et al. 1975
(female)	1009	Zakhari et al. 1978, citing data of Lee et al. 1975
(both sexes)	660	Dilley et al. 1978a, 1982a
Rat (male)	1320	Dilley et al. 1978a, 1982a
(male)	1010	Zakhari et al. 1978, citing data of Lee et al. 1975
(female)	794	Dilley et al. 1978a, 1982a
(female)	820	Zakhari et al. 1978, citing data of Lee et al. 1975

urine is due to a metabolite of TNT. Although acute human exposures are more likely to take place during the manufacturing process where skin absorption and inhalation are the primary routes of intake, little information on administration to laboratory animals by these routes was found. Two cutaneous doses of 2000 mg/kg in 8 hours to a cat resulted in death of the animal (Dacre and Rosenblatt 1974, citing data of von Oettingen 1941). A dose of 300 mg/kg produced no effects.

Subacute oral toxicity studies with rats, mice, and dogs resulted in depressed body weight, reduced food consumption, mild to moderate hemolytic anemia, enlarged spleens and livers, hemosiderosis of the spleen, and colored urine. Recent studies are summarized in Table 7.5. In dogs and rats increased cholesterol (and possibly bilirubin) and decreased serum glutamic-pyruvic transaminase (SGPT) levels were observed. At high dose levels (125 mg/kg/day or greater) testicular atrophy, increases in kidney weights with deposition of pigment, lymphocytosis, elevated methemoglobin levels, and cerebellar lesions were observed in rats. The only neurological symptom was inactivity in dogs. Except for testicular atrophy, the toxic effects were reversible in rats allowed a 4-week recovery period. No-observable-effect levels in these 90-day studies were: dogs, 0.20; rats, 1.40; and mice, 7.46 mg/kg/day (Dilley et al. 1978a).

Inhalation (insufflation) of doses of 25 or 50 mg/kg, 6 days/week for 17 weeks resulted in a 33 percent mortality rate in dogs (Dacre and Rosenblatt 1974, citing data of von Oettingen et al. 1944). Effects included salivation, vomiting, diarrhea, incoordination, weakness, and moderate anemia. No cyanosis was evident.

TABLE 7.5. SUBACUTE (90-DAY) ORAL TOXICITY STUDIES WITH TNT

Species	Dose	Effects	Reference
Rat	0.002% in diet[a]	No effects	Dilley et al. 1978a
	0.01% in diet	Red urine (TNT metabolite), slight anemia	
	0.05% in diet	Body weights and food intake depressed, anemia, low serum iron, enlarged spleens (males), hemosiderosis of spleen, increased liver weights (female), red urine	
	0.25% in diet	Body weight and food intake depressed, enlarged spleens with hemosiderosis, testicular atrophy, enlarged livers, smaller kidneys, anemia, leukocytosis, increased uric acid level, decreased SGPT[c], increased cholesterol	
Rat	1 mg/kg/day	No effects	Levine et al. 1981a
	5 mg/kg/day	Slight decrease in body weight gain (males)	
	25 mg/kg/day	Reduced food intake, hypercholesterolemia, anemia	
	125 mg/kg/day	Reduced food intake, hypercholesterolemia, anemia, enlarged spleens with congestion and hemosiderosis, enlarged livers with hepatocellular hypertrophy, testicular atrophy with degeneration of the seminiferous tubules, slight increase in kidney weight	
	300 mg/kg/day	Same as at 125 mg/kg/day; in addition, elevated methemoglobin levels and cerebellar lesions	
Mouse	0.001% and 0.005% in diet[b]	No alteration in any parameter measured	Dilley et al. 1978a
	0.025% in diet	Temporary decrease in initial body weight and food intake, red urine, hemosiderosis in spleen	
	0.125% in diet	Temporary initial body weight depression and anemia, enlarged spleens with hemosiderosis, enlarged livers with occasional necrosis	
Dog	0.20 mg/kg/day	No detectable effects in any parameter measured	Dilley et al. 1978a
	2.0 mg/kg/day	Possible depression in body weight gain, decreased serum iron, enlarged kidneys with lymphocyte deposition	
	20 mg/kg/day	Decreased body weight gain and food intake, increased liver, spleen, and possibly adrenal weights, mild to moderate anemia, decreased PMN[d], increased cholesterol and bilirubin, decreased SGPT and iron, red urine, occasional inactivity and nystagmus	
	0.02, 0.1, or 1 mg/kg/day	No signs of toxicity other than temporary episodes of emesis to which a tolerance developed	Hart 1974

a. Calculated average daily intake was 1.40, 6.97, 34.7, and 160 mg/kg for males and 1.45, 7.41, 36.4, and 164 mg/kg for females at 0.0025, 0.01, 0.05, and 0.25% TNT diets, respectively (Dilley et al. 1982a).

b. Calculated average daily intake was 1.56, 7.46, 35.7, and 193 mg/kg for males and 1.57, 8.06, 37.8, and 188 mg/kg for females at 0.001, 0.005, 0.025, and 0.125% TNT diets, respectively.

c. SGPT = serum glutamic-pyruvic transaminase.

d. PMN = polymorphonuclear leukocytes.

7.1.2.2 Mutagenicity

The Ames assay system, using histidine-requiring strains of Salmonella typhimurium, was used to determine the mutagenicity of explosive grade TNT (Won et al. 1976). In pour-plate tests, at concentrations of 0.5 to 10 µg TNT per mL agar overlay, unactivated TNT was a frameshift mutagen, characterized by a linear mutagenic response curve. The major microbial metabolites of TNT appeared to be nonmutagenic. In further studies TNT dissolved in DMSO was mutagenic to three additional strains of Salmonella, both in the presence and absence of metabolic activation (Dilley et al. 1978a; Ellis et al. 1978). Mutagenicity was, however, reduced by the metabolic activation system. It should be noted that the Salmonella strains used have endogenous aromatic nitro reductase enzymes.

TNT caused chromosomal changes in the tibial bone marrow cells of rats exposed chronically by topical application (Zakhari et al. 1978, citing data of Geshev and Kincheva 1974). In contrast, no genetic damage was observed in bone marrow cells from rats fed TNT (Dilley et al. 1978a). TNT gave a positive response in the unscheduled DNA synthesis assay when no metabolic activation was used (Dilley et al. 1978a).

7.1.2.3 Carcinogenicity

A review of results of chronic studies indicates that no tumors attributable to TNT have been found in experimental animals.

7.1.2.4 Teratogenicity and Reproductive Effects

No studies on the teratogenicity of TNT have been reported. Irreversible testicular atrophy was produced in rats fed at a dose level of 0.25 percent in their diets for 13 weeks (Dilley et al. 1982a).

7.1.3 Recommendations and Standards

Based primarily on the study of Morton et al. (Section 7.1.1), the American Conference of Governmental Industrial Hygienists (ACGIH) lowered the recommended threshold limit value (TLV) for skin exposures in the workplace from 1.5 mg/m^3 to the present time weighted average (TWA) of 0.5 mg/m^3 (ACGIH 1983). The short term exposure limit (STEL) is 3 mg/m^3. The Occupational Safety and Health Administration (OSHA 1982) has a standard of 1.5 mg/m^3 for skin exposure. The U.S. Army has a standard of 0.5 mg/m^3 (DARCOM Regulation 40-3, as reviewed in Rosenblatt 1980).

7.2 RDX

RDX (hexahydro-1,3,5-trinitro-1,3,5-triazine) is a white crystalline high explosive, extensively used by the military. Inhalation of dust at manufacturing facilities and accidental ingestion by field personnel have resulted in epilepticlike seizures with loss of consciousness. While the potential for acute exposure at manufacturing and handling

facilities has been controlled by industrial hygiene procedures, the potential for chronic exposures exists in these facilities and in areas near manufacturing and demilitarization sites where RDX may be discharged to local waterways and eventually reach potable groundwater. RDX has been identified as one of the principal constituents of wastewater discharge from munitions production and handling facilities. After demilitarization, RDX has been stored in open pits from where it may leach into surface and groundwater.

7.2.1 Health Effects in Humans

Occupationally related cases of RDX intoxication in the munitions industry have been documented in several studies. In addition, military field personnel have occasionally suffered symptoms of RDX intoxication following ingestion of RDX-containing plastic explosives or during their use as a cooking fuel. RDX exerts its primary toxic effect on the central nervous system.

In the munitions industry, RDX exposure has occurred from inhalation; there is little evidence of skin absorption (Rosenblatt 1980). Chronic intoxication in workers is characterized by epileptiform seizures (generalized convulsions) and unconsciousness (Stokinger et al. 1982a). Convulsions may appear without warning or be preceded by one or two days of insomnia, restlessness, and irritability. Seizures are followed by temporary amnesia, disorientation, and asthenia.

Epileptiform seizures have occurred in workers manufacturing RDX in Italy (Barsotti and Crotti 1949), Germany (Vogel 1952), and the USSR (Sklyanskaya and Pozhariskii 1944). Seizures occurred most frequently in workers handling RDX in a powdered form. In most cases, there was complete recovery when workers were removed from the source of exposure.

Zaplan et al. (1965) described five cases of illness among 26 workers engaged in pelletizing RDX in an explosives plant in the United States. Exposure occurred from the release of dust in the workroom air during dumping of dried RDX powder in an unventilated room or while sweeping the RDX that had spilled onto the floor. Either at work or several hours after returning home, the worker had a convulsion or became unconscious without a convulsion. Presymptoms and postsymptoms were similar to those described above. No abnormal physical findings except those relating to the central nervous system were noted. Recovery was complete with no sequelae. When control measures were installed in the plant, the illnesses disappeared.

Similar evidence of systemic intoxication was not observed at the Holston Ordnance Works during World War II (Sunderman 1944). In this operation the Bachmann process, which requires an essentially closed system for the reaction mixture, was used. Standard procedures did not involve the handling of dry RDX. However, primary irritation and sensitization dermatitis, particularly of the face and eyelids, occurred in workers exposed to fumes during the nitration process. Studies with volunteers indicated that an unidentified component in the fumes from

the reaction mixture was responsible. Patch testing with solid RDX failed to produce any local skin lesions similar to those observed during the manufacture of RDX (Sunderman 1944; von Oettingen et al. 1949).

In the 1970s a cross-sectional epidemiologic study at five U.S. Army ammunitions plants was conducted in order to identify adverse health effects among workers with RDX exposures (Hathaway and Buck 1977). In particular, the investigators attempted to identify abnormalities of the hematologic, hepatic, and renal systems, and the presence of autoimmune disease. Results of the study showed no excess of autoimmune disease and no statistically significant differences in abnormalities of the hematologic, hepatic, or renal systems in employees with 8-hour time weighted exposures to RDX of up to 1.57 mg/m^3 (0.28 mg/m^3 average) compared with unexposed controls.

Toxic symptoms have been observed in field personnel following the intentional or accidental ingestion of composition C-4, a plastic explosive containing 91 percent RDX, 2.1 percent polyisobutylene, 1.6 percent motor oil, and 5.3 percent di-(2 ethylhexyl) sebacate (Stone et al. 1969). C-4 may also be used as a field cooking fuel when other sources of heat are unavailable, resulting in inhalation of fumes. Acute effects were seen within a few hours after exposure (Stone et al. 1969).

Symptoms in six cases which required hospitalization are as follows. Ingestion was followed within a few hours by multiple generalized seizures, hematuria, severe nausea and vomiting, muscle twitching, and mentation changes. Abnormal laboratory findings included neutrophilic leukocytosis, elevated serum glutamic oxalacetic transaminase (SGOT), elevated blood urea nitrogen (BUN), proteinuria, and hematuria. Liver biopsies appeared normal. Anemia and loss of memory for recent events persisted for one month (time of discharge) in a patient who had ingested the largest dose, approximately 180 g of C-4 explosive. No fatalities were reported.

7.2.2 Toxic Effects in Laboratory Animals

7.2.2.1 General Toxicity

As indicated by the values in Table 7.6, RDX is moderately to acutely toxic when administered orally to laboratory animals. Comparison of these values with those for TNT (Table 7.4) shows that RDX is more toxic after single oral exposures. As would be expected, the intravenous LD_{50} of RDX is much lower than the oral LD_{50}.

Oral LD_{50} values for the same species may differ among laboratories because the acute LD_{50} is dependent on the physical form of the RDX and on the method used to suspend or dissolve it (Schneider et al. 1977). Coarse, granulated powder produced an oral LD_{50} of 300 mg/kg in rats; finely powdered RDX in saline slurry produced the same LD_{50} value as when dissolved in DMSO, approximately 100 mg/kg. Differences in toxicities of the preparations were reflected in plasma RDX concentrations, the value following administration of the coarse powder being lower than when administered in the other preparations.

TABLE 7.6. RDX LETHALITY DATA

Species	Route/Vehicle	LD_{50} (except as otherwise noted)	Reference
Rat	Oral/gum acacia	~200 mg/kg	von Oettingen et al. 1949
	Oral/corn oil	~70 mg/kg	Dilley et al. 1978a
	Oral/DMSO or saline slurry	~100 mg/kg	Schneider et al. 1977
	Oral, coarse powder	300 mg/kg	Schneider et al. 1977
	Oral/methyl cellulose-polysorbate 80	118 mg/kg	Cholakis et al. 1980
	Oral/food	LD_{LO}: 75 mg/kg	Sunderman 1944
Mouse	Oral/linseed oil	500 mg/kg	Lewis and Tatkin 1982, citing data of Sklyanskaya and Pozhariskii 1944
	Oral/corn oil	<75 mg/kg (males) 86 mg/kg (females)	Dilley et al. 1978a
	Oral/methyl cellulose-polysorbate 80	80 mg/kg	Cholakis et al. 1980
	Intravenous/DMSO	19 mg/kg	McNamara et al. 1974
Cat	Oral/linseed oil	LD_{LO}: 100 mg/kg	Lewis and Tatkin 1982, citing data of Sklyanskaya and Pozhariskii 1944
Rabbit	Oral/linseed oil	LD_{LO}: 500 mg/kg	Lewis and Tatkin 1982, citing data of Sklyanskaya and Pozhariskii 1944
Guinea pig	Intravenous/DMSO	25 mg/kg	McNamara et al. 1974
Dog	Intravenous/DMSO	LD_{100}: 40 mg/kg	McNamara et al. 1974

As in man, central nervous system excitation is the most prominent acute effect of RDX on most animals. Other toxic symptoms include gasping and labored breathing. In rats administered acute lethal oral doses, the majority of deaths occurred within 24 hours (von Oettingen et al. 1949). At autopsy there was moderate to marked congestion of the gastrointestinal tract and lungs in some animals. There were no pathologic changes in the brain, although nonspecific lesions were observed in the renal tubules, liver, and the heart muscle.

Other toxicity studies, some of them directed toward determining a no-effect dose level and a water quality criterion, are summarized in Table 7.7. In 90-day subchronic toxicity studies, oral doses of 28 mg/kg were not toxic to rats; however, in one of the studies deaths resulted from exacerbation of chronic respiratory disease (Schneider et al. 1978). Oral doses of 10 mg/kg or less resulted in no signs of toxicity except for temporary episodes of emesis in dogs (Hart 1974). Incorporation of RDX in the diets of rats at a level of 10 mg/kg over a two-year period resulted in no evidence of toxicity (Hart 1976). Rats allowed free access to RDX-saturated drinking water (50 to 70 µg/mL representing a daily dose of 5 to 8 mg/kg) for up to 90 days displayed no overt signs of toxicity, and all organs appeared normal at necropsy (Schneider et al. 1978).

In the topical administration studies summarized in Table 7.7, McNamara et al. (1974) state in their summary that "RDX in the three solvents did not penetrate the skin, as evidenced by the lack of physiological responses in dogs and unchanged blood component values in rabbits." Paralysis and deaths did, however, occur in rabbits following topical application of large doses. One rabbit suffered paralysis after five 1.0-mL doses of RDX (33 percent in DMSO), and three rabbits died, one after the fifth 0.1-mL dose, one after the tenth 1.0-mL dose of 5.4 percent RDX in acetone, and one after the eighth 1.0-mL application of 7.5 percent RDX in cyclohexanone. The authors do caution against skin contact with RDX in the solvents, partly because the solvents themselves cause skin and ocular effects. This study, although inconclusive, is particularly relevant because of the use of these solvents for recrystallization of RDX at production facilities (Rothrock et al. 1981). The paralysis and deaths indicate that when administered in solvents at high doses, some of the compound enters the body. Additional studies using radiolabeled compounds for topical administration could lead to more conclusive results.

Topical application of RDX in DMSO, acetone, or cyclohexane to rabbits and guinea pigs produced no irritation greater than that from the solvents alone and gave no evidence of sensitization (McNamara et al. 1974). Intradermal injection of RDX in the solvents caused severe skin damage.

RDX produces nervous system toxicity. Despite the presence of the NO_2 moiety, RDX does not have the characteristic nitrite actions of lowering blood pressure through vasodilation or of forming methemoglobin (von Oettingen et al. 1949, Schneider et al. 1978). The short time

TABLE 7.7. SUMMARY OF RDX SUBACUTE AND CHRONIC TOXICITY STUDIES

Species	Route/dose	Effects	Reference
Rat	Oral-25, 50, 100 mg/kg for 10 weeks	Weight loss, hyperactivity, convulsions, 40-87% mortality; congestion of lungs and GI tract	von Oettingen et al. 1949
	Oral-15 mg/kg for 10 weeks	No toxic symptoms or death	von Oettingen et al. 1949
	Oral-300 or 600 mg/kg for up to 13 weeks	Mortality in less than 13 weeks	Levine et al. 1981a, 1981b
	Oral-1, 10, 30, or 100 mg/kg for 13 weeks	Hyperactivity to approach, convulsions, dose-related reductions in body weight gain, decreased serum triglycerides, marginal leukocytosis	Levine et al. 1981a, 1981b
	Oral-40 mg/kg for 90 days	Decreased weight gain in males related to decreased food consumption; no behavioral, clinical chemistry, or organ pathology changes	Cholakis et al. 1980
	Oral-28 mg/kg for 90 days	No effects	Cholakis et al. 1980
	Oral-20 mg/kg for 90 days	Lethargy, weight loss, rough coat hair; no neurological symptoms, death from exacerbation of chronic respiratory disease	Schneider et al. 1978
	Oral-1.0, 3.1, or 10 mg/kg for two years	No evidence of toxicity	Hart 1976
	Oral (drinking water)- 5 to 8 mg/kg for 90 days	No signs of toxicity	Schneider et al. 1978
Mouse	Oral-10, 14, 20, 28, or 40 mg/kg for 90 days	No effects	Cholakis et al. 1980
	Oral-320 m/kg for 90 days	Hyperactivity, mortality, increased liver and kidney weight, hepatocellular vacuolization, tubular nephrosis	Cholakis et al. 1980
Dog	Oral-50 mg/kg for 6 weeks	Weight loss, hyperactivity, hyperirritability, convulsions; no blood or tissue effects	von Oettingen et al. 1949
	Oral-0.1, 1.0, or 10 mg/kg for 90 days	Temporary episodes of emesis	Hart 1974
	Topical-480 mg/kg in DMSO on three consecutive days	No significant changes in blood pressure, heart rate, EKG or EEG patterns, or respiration	McNamara et al. 1974
Rabbit	Topical-0.1 or 1.0 ml in DMSO (33% wt/vol), 5 d/wk for 4 weeks (330-3300 mg/kg)	No systemic effects or cataracts; dermatitis	McNamara et al. 1974
	Topical-0.1 or 1.0 ml in acetone (5.4% wt/vol) 5d/wk for 4 weeks (54-540 mg/kg	No organ or tissue lesions; two deaths	McNamara et al. 1974
	Topical-0.1 or 1.0 ml in cyclohexanone (7.5% wt/vol) 5 d/wk for 4 weeks (75-750 mg/kg)	No organ or tissue lesions; one death	McNamara et al. 1974
Guinea pig	Topical-316 (1 application) to 1,000 (3 applications) mg/kg in DMSO	No effects to slight erythema; apprehension and weight loss at highest dose	McNamara et al. 1974

period between intravenous injection of RDX to rats and dogs and the onset of convulsions suggests that the parent compound is responsible for the central nervous system effects (Schneider et al. 1977, McNamara et al. 1974).

7.2.2.2 Mutagenicity

Results of mutagenicity tests with RDX were all negative. Using five strains of histidine-requiring Salmonella and concentrations of 1.25 and 0.625 mg per spot, no reversions were formed (Whong et al. 1980). In the plate assay with induced rat liver S-9 activation and at doses up to 2.5 mg per plate, RDX gave negative results with all five strains of Salmonella (Cholakis et al. 1980, Whong et al. 1980). RDX was also not mutagenic in the rat dominant lethal mutation test at doses up to 50 mg/kg/day (Cholakis et al. 1980).

Unscheduled DNA synthesis (UDS) assays were also negative (Dilley et al. 1978a). At a maximum test concentration of 4,000 µg/mL (based on the solubility of RDX) UDS was not observed with or without metabolic activation.

7.2.2.3 Carcinogenicity

Only one study on the potential carcinogenicity of RDX was located. Although this was not a definitive study, histological examinations at the conclusion of a two-year feeding study with rats at a dose level of 10 mg/kg/day revealed no significant incidence of neoplasms in 16 tissues examined (Hart 1976).

7.2.2.4 Teratogenicity and Reproductive Effects

In one- and two-generation reproduction studies, RDX did not cause reproductive effects in rats or rabbits at doses of 20 mg/kg/day, nor did it cause teratogenic effects (Cholakis et al. 1980). At this dose RDX did produce severe maternal toxicity including some deaths and embryotoxicity in rats. Reproductive effects, however, were noticed in males and females receiving 50 mg/kg/day. These effects included a reduced number of pregnancies and a poor survival of offspring from the pregnancies and were attributed to poor nutrition resulting from the general toxicity.

7.2.3 Recommendations and Standards

The threshold limit value (TLV) for RDX recommended by the American Conference of Governmental Industrial Hygienists (ACGIH 1983) is 1.5 mg/m^3 for skin exposure. The value was based on the analogy of RDX to TNT and the demonstrated effectiveness in the prevention of injury at an Atomic Energy Commission establishment when RDX was maintained below 1.5 mg/m^3 (Stokinger et al. 1982a, citing data of Hyatt and Milligan 1953). The short-term exposure limit (STEL) is 3 mg/m^3.

Based on their subchronic (drinking water) studies, Schneider et al. (1978) suggest an ingestion limit of 0.1 mg/kg/day or 2 to 3 ppm in potable water.

7.3 HMX

HMX (octahydro-1,3,5,7-tetranitro-1,3,5,7-tetrazine) is a military unique compound, which, like RDX, is produced exclusively by Holston Army Ammunitions Plant in Kingsport, Tennessee, where it is present in the wastewater discharge. It is also present at RDX LAP facilities, since RDX (Type B) contains approximately 5 percent (3 to 10 percent) HMX (Kitchens et al. 1979). At Army arsenals, plant personnel may handle HMX dissolved in various solvents. Little information on acute and chronic exposures to HMX is available.

7.3.1 Health Effects in Humans

Two occupational exposure studies were reported in the literature. Hathaway and Buck (1977) studied the effects of exposures to RDX on workers at an Army munitions plant. RDX concentrations as high as 1.5 mg/m^3 were measured in the air. HMX was presumably present in the air, but was not measured. No differences in the number of abnormalities of the hematologic, hepatic, or renal systems, or in the presence of autoimmune disease were found among the workers as compared to unexposed controls.

Health hazards to workers at the Holston Plant during the manufacture of RDX have been discussed (Section 7.2.1). No occupational monitoring studies are available from the Holston Plant. Using human volunteers from the Holston plant, patch testing with solid HMX indicated that this compound is a skin irritant (Sunderman 1944).

7.3.2 Toxic Effects in Laboratory Animals

7.3.2.1 General Toxicity

Studies on the acute (oral and intravenous administration) and subacute (topical administration) toxicity of HMX are summarized in Tables 7.8 and 7.9, respectively. Because of the low water solubility of HMX, most toxicity studies involve the administration of this compound in solvents such as dimethylsulfoxide (DMSO). Although no oral LD_{50} value for rats was determined, 100 mg/kg killed three out of 10 animals in 40 days (Kagan et al. 1975). HMX appears to be less toxic than RDX.

HMX exerts a nitritelike effect on the cardiovascular system. The immediate effect of intravenous administration is circulatory system collapse with delayed central nervous system disturbances of hyperactivity and convulsions (McNamara et al. 1974). Death occurred within 5 minutes after injection to rats and guinea pigs. Dogs given acute doses showed changes in blood pressure, heart rate, respiratory rate, EKG, and EEG patterns. However, methemoglobin formation, another toxic effect typical of nitrite exposure, was not observed in the dogs.

TABLE 7.8. ACUTE LD_{50} VALUES FOR HMX

Species	Route/Vehicle	LD_{50}	Reference
Mouse	Intravenous/DMSO	28.9 mg/kg	McNamara et al. 1974
	Oral	1,500 mg/kg	Kagan et al. 1975
Guinea pig	Intravenous/DMSO	28.2 mg/kg	McNamara et al. 1974
	Oral	300 mg/kg	Kagan et al. 1975

In a subacute study, HMX was administered topically in three different solvents; toxic effects were produced only at high doses (see Table 7.9) (McNamara et al. 1974). When applied repeatedly as a 33 percent solution in DMSO, deaths were recorded in rabbits and guinea pigs. Rabbits died after repeated 1.0-mL doses and guinea pigs in 24 to 48 hours after single doses of 465, 477, 507, and 546 mg/kg. In surviving rabbits, blood component values were unchanged, and none of the physiological responses seen in the acute studies with dogs were observed. Again methemoglobin was not formed. Lesions were not found in the liver, kidney, spleen, lung, trachea, heart, intestine, bladder, muscle, bone, or bone marrow of rabbits which died or were sacrificed following repeated topical applications of HMX in the three solvents. Gross examination of eyes revealed no cataracts.

Topical or intradermal application of DMSO, acetone, or cyclohexanone or of HMX in these three solvents 3 days a week for 3 weeks followed in 2 weeks by topical or intradermal challenge, gave no evidence of sensitization (McNamara et al. 1974). Ocular administration showed that HMX was no more damaging to the eyes than the solvents alone, but the solvents themselves caused cataracts in guinea pigs.

7.3.2.2 Mutagenicity

HMX was not mutagenic in the Ames Salmonella spot test or plate incorporation assay (Whong et al. 1980). In five strains of Salmonella, no histidine-requiring reversions were formed with or without metabolic activation.

The Ames mutagenicity assay was also performed on the effluent from a pilot wastewater treatment facility at a munitions plant (Kitchens et al. 1979, citing data of Stilwell et al. 1977). Before treatment the wastewaters contained HMX and RDX levels ranging from approximately 0.1 to 5.0 ppm. The treated wastewater had HMX and RDX levels ranging from less than 0.05 to 0.7 ppm. Results indicated that the wastewater, both before and after treatment, had no mutagenic activity.

7.3.2.3 Carcinogenicity

No studies have been conducted to determine the carcinogenic effects of HMX.

TABLE 7.9. EFFECTS OF HMX ADMINISTERED TOPICALLY IN VARIOUS SOLVENTS[a]

Species	Dose/Exposure	Effect
Rabbit	0.1 to 1.0 mL in DMSO (33 percent wt/vol) 5 days/week for 4 weeks (330 to 3,300 mg/kg)	No systemic effects (unchanged blood components); mild desquamation of skin; three deaths
	0.1 to 1.0 mL in acetone 2.0 percent wt/vol) 5 days/week for 4 weeks (20 to 200 mg/kg)	No systemic effects; no cutaneous irritation
	0.1 to 1.0 mL in cyclohexanone (2.5 percent wt/vol) 5 days/week for 4 weeks (25 to 250 mg/kg)	No cutaneous or systemic toxicity; posterior leg paralysis
Guinea pig	Single and repeated 0.5-mL applications of 33 percent HMX in DMSO	7/12 animals - no symptoms; 5/12 animals - deaths after single doses of 465, 477, 507, and 546 mg/kg and two doses of 1,126 m/kg
	Single applications of 316 to 2,000 mg/kg in DMSO	No deaths
	Three 1,000 mg/kg doses in DMSO	Apprehension, weight loss, skin effects
Dog	17.5 or 289 mg/kg in DMSO or acetone 5 days/week for 4 weeks	No changes in blood pressure, heart rate, or respiratory rate
	480 mg/kg in DMSO on three consecutive days	No gross effects

a. Data from McNamara et al. 1974.

7.3.2.4 Teratogenicity and Reproductive Effects

No studies have been conducted to determine the teratogenic and reproductive effects of HMX.

7.3.3 Recommendations and Standards

There are no industrial hygiene standards for HMX. Based on the lowest single percutaneous dose to cause death in guinea pigs, 465 mg/kg, the lethal dose for a 70-kg man would be approximately 33 g of HMX administered in 100 mL of DMSO (McNamara et al. 1974).

7.4 BY-PRODUCTS OF TNT, RDX, AND HMX MANUFACTURE

7.4.1 NO_x

Exposure of TNT workers to fumes in nitrating rooms where toluene is mixed with sulfuric and nitric acids can cause severe bronchitis and edema of the lungs (Zakhari et al. 1978). These fumes consist of nitric acid fumes, nitrogen oxides (NO_x), methane, hydrogen, and chlorine gases.

In the respiratory tract, nitrogen oxides form nitric and nitrous acids (Sax 1979). The acids formed are irritating, causing congestion of the throat and bronchi and edema of the lungs. High concentrations of these fumes, 200 to 700 ppm, may be fatal. Continued exposure to low concentrations of the fumes, insufficient to cause pulmonary edema, may result in chronic irritation of the respiratory tract, with cough, headache, loss of appetite, dyspepsia, corrosion of the teeth, and gradual loss of strength.

The recommended threshold limit value (TWA) for nitrogen dioxide (NO_2) is 3 ppm or 6 mg/m^3; the STEL is 5 ppm or 10 mg/m^3 (ACGIH 1983). For nitric oxide (NO) the TWA threshold limit value is 25 ppm (30 mg/m^3); the STEL value is 35 ppm (45 mg/m^3) (ACGIH 1983). The Occupational Safety and Health Administration lists a ceiling value (a value which at no time should be exceeded) of 5 ppm (9 mg/m^3) for nitrogen dioxide (OSHA 1982). The OSHA standard (TWA) for nitric oxide is 25 ppm (30 mg/m^3).

7.4.2 Tetranitromethane

Tetranitromethane (TNM) is a volatile by-product of the manufacture of TNT and may be present in crude TNT to the extent of 0.12 percent (Sievers et al. 1947, citing data of Moore 1917). It has a sufficiently high vapor pressure (13 mm Hg at 25°C) to present an inhalation hazard (Rosenblatt 1980). It is probably not absorbed through the skin (Machle et al. 1940). Purification processes such as sulfitization remove practically all TNM (Sievers et al. 1947). Thus, in present day operations, TNM would present a potential health hazard only prior to purification processes and during clean-up and repair operations.

7.4.2.1 Health Effects in Humans

During the early part of World War I, numerous cases of TNT poisoning occurred that were attributed to the TNM content of the TNT (Sievers et al. 1947). The irritative property of TNM fumes constituted an industrial nuisance and at times limited the efficiency of workers during manufacturing. Workers experienced nasal irritation, burning of the eyes, dyspnea, coughing, chest oppression, and dizziness. Continued or moderately heavy exposure led to drowsiness, headache, anemia, cyanosis with respiratory distress, and bradycardia. Severe intoxications and deaths have occurred during the handling of crude heated TNT (Sievers et al. 1947, citing data of Koelsch 1917). Symptoms included cyanosis, respiratory distress, pulmonary edema, methemoglobinemia, and unconsciousness. Levels of TNM in these early cases are unknown.

Symptoms experienced during the laboratory production of TNM were similar to those above but less severe (Hager 1949). Sensitivity among the workers varied greatly. In addition to mucous membrane and respiratory passage irritation, chronic symptoms included headaches and respiratory distress. Skin irritation did not occur.

Sax (1979) states that prolonged exposure to vapors causes damage to liver, kidney, and other organs, but the source of this information is not given.

7.4.2.2 Toxic Effects in Laboratory Animals

TNM is highly toxic via the inhalation route. A summary of data on the response of animals to inhalation of various concentrations of TNM appears in Table 7.10. It should be noted that in the studies by Sievers et al. (1947) animals were exposed to fumes from crude TNT, and, although TNM concentrations were sampled and analyzed, other contaminants could have been present. Inhalation exposures produced LC_{50} values of 17.5 ppm for rats and 54.5 to 75 ppm for mice (Table 7.10). By oral and intravenous routes of administration, LD_{50} values were, for rats, 130 mg/kg and 12.6 mg/kg and, for mice, 375 mg/kg and 63.1 mg/kg, respectively (Kinkead et al. 1977).

TNM exhibits two types of acute toxic effects depending on the mode of administration (Kinkead et al. 1977). In the inhalation studies, exposed animals exhibited similar symptoms, chiefly those of respiratory tract irritation and pulmonary edema. Death was due to severe pneumonia. Methemoglobinemia was rarely present. In contrast, the acute toxic response following oral and intravenous administration was characteristic of acute methemoglobinemia.

The toxic effects of TNM were for a time thought to be the same as those for nitrogen dioxide and were attributed to a possible breakdown of TNM to that compound. Although TNM and NO_2 produce similar symptoms during inhalation experiments, methemoglobinemia is rarely present in TNM-exposed animals. Also, the magnitude of effects of the two compounds are not equivalent on a 4:1 ($C(NO_2)_4$:NO_2) atom count basis (Kinkead et al. 1977). At this time the mechanism of TNM intoxication is not clear, and further studies are needed.

TABLE 7.10. RESPONSE TO VARIOUS ATMOSPHERIC CONCENTRATIONS OF TETRANITROMETHANE (TNM)[a]

Animal	Number of animals tested	Concentration (ppm)	Duration of exposure	Response	Reference
Cat	1	100	20 min	Death in 1 hr	Stokinger 1982a, citing data of Flury and Zernik 1931
	1	10	20 min	Death in 10 days	Stokinger 1982a, citing data of Flury and Zernik 1931
	5	7-25	2 ⅘-5 hr	Death in 1-5 ⅓ hr	Sievers et al. 1947
	2	3-9	6 hr x 3	Severe irritation	Sievers et al. 1947
	2	0.1-0.4	6 hr x 2	Mild irritation	Sievers et al. 1947
Rat	20	1,230	1 hr	All died in 25-50 min	Horn 1954
	20	300	1 ½ hr	All died in 40-90 min	Horn 1954
	20	33	10 hr	All died in 3-10 hr	Horn 1954
	19	6.35	6 months	11 deaths	Horn 1954
		17.5	4 hr	LC_{50}	Kinkead et al. 1977
		0.4	1 hr	Effects on conditioned reflex	Kinkead et al. 1977, citing data of Korbakova 1960
	100	7.5	14 days	65 deaths	Kinkead et al. 1977
	100	5.0	14 days	16 deaths	Kinkead et al. 1977
	100	3.5	14 days	Decrease in body weight, increase in lung weight	Kinkead et al. 1977
Dog	2	6.35	6 months	Mild symptoms	Horn 1954
Mouse		54.5	4 hr	LC_{50}	Kinkead et al. 1977
		75	2 hr	LC_{50}	Kinkead et al. 1977, citing data of Korbakova 1960

a. Modified from Stokinger 1982a.

TNM has been characterized as a mutagen through the use of the Ames test. Dissolved in DMSO and with metabolic activation, it was mutagenic to two strains of Salmonella at 30 µg per plate (Ellis et al. 1978). At concentrations above 1.0 µg per plate, it was bactericidal without metabolic activation. Additional tests in mammalian systems would be desirable.

TNM is currently being tested for carcinogenic activity in Fischer rats under the National Toxicology long-term carcinogenesis bioassay program (Whitmire 1981). The inhalation route of administration is being used.

7.4.2.3 Recommendations and Standards

The American Conference of Governmental Industrial Hygienists (ACGIH 1983) suggests a threshold limit value (TLV) of 1 ppm (8 mg/m^3) for industrial workers. The Occupational Safety and Health Administration standard is also 1 ppm (OSHA 1982). Although human experience with measured concentrations is lacking, results of the animal experiments indicate an irritative response at 0.1 to 0.4 ppm and effects on body and lung weight at 3.5 ppm.

7.4.3 Trinitromethane

Trinitromethane (nitroform) is not formed as a by-product in the manufacture of TNT, but tetranitromethane (TNM) is. At one time, TNM was vented to the atmosphere, but it now is scrubbed with aqueous sodium carbonate containing stabilized hydrogen peroxide which converts the TNM to trinitromethane.

7.4.3.1 Health Effects in Humans

Little published information on the health effects of trinitromethane was found. This compound is somewhat irritating to the skin, eyes, and respiratory tract (Sax 1979). Inhalation can cause headache, nausea, and mild narcosis.

7.4.3.2 Toxic Effects in Laboratory Animals

Toxic concentrations in air for mice are: LD_{50}, 0.8 and LD_{100}, 1.0 mg/L (Gilbert 1980, citing data of Timofieveskaya 1964). Trinitromethane is moderately toxic following intraperitoneal injection. The reported LD_{50} value in mice was 115 mg/kg (Lewis and Tatken 1982).

7.4.3.3 Recommendations and Standards

There are no industrial hygiene standards for trinitromethane.

7.4.4 Methyl Nitrate

Methyl nitrate is a volatile by-product of RDX and HMX manufacture. It appears to be of little or no commercial interest. Little information on human health effects and toxicity is available.

7.4.4.1 Health Effects in Humans

No injuries to workers from exposures to methyl nitrate have been reported in the published literature. In tests conducted on two human subjects, inhalation of minimal doses of 117 and 417 mg, respectively, was required to induce headaches (von Oettingen 1946).

7.4.4.2 Toxic Effects in Laboratory Animals

LD_{50} values for several animal species are listed in Table 7.11. Methyl nitrate appears to be moderately toxic via oral and inhalation routes of administration. Responses of laboratory animals to acute doses of methyl nitrate by inhalation were dose-related and followed a general pattern of lethargy, decreased respiratory rate, and cyanosis (Kinkead et al. 1977). All animals were inactive during the 4-hour exposure period. The toxicity of methyl nitrate was much greater in rats than in mice, and rats that died as a result of inhalation died either during exposure or in the following 12 hours, while mouse deaths were often delayed from 3 to 11 days postexposure.

At high oral dose levels, rats exhibited labored breathing and gasping. Results of pathological examinations revealed mild to moderate pulmonary congestion with focal areas of hemorrhage and methemoglobinemia. Animals surviving a 14-day postexposure period appeared to recover completely. The action of methyl nitrate is characteristic of the aliphatic nitrates: dilation of blood vessels and methemoglobin formation (Stokinger 1982a). The vascular dilation accounts for the lowering of blood pressure and headache, the latter being the main symptom produced in humans. Methemoglobin formation indicates the in vivo formation of nitrite.

Methyl nitrate was reported to have mutagenic effects on Escherichia coli bacteriophage T4B at 0.54-M concentration (Rosenblatt 1980).

No studies on carcinogenicity or teratogenicity were located.

7.4.4.3 Recommendations and Standards

No industrial hygiene standards for methyl nitrate have been established.

TABLE 7.11. ACUTE TOXICITY OF METHYL NITRATE[a]

Species	Route	LD_{50}
Rat	Oral	344 mg/kg
	Inhalation	1,275 ppm/4 hr
Mouse	Oral	1,820 mg/kg
	Inhalation	5,942 ppm/4 hr
Guinea pig	Oral	548 mg/kg

a. From Kinkead et al. 1977.

7.4.5 TNT Isomers

In the TNT manufacturing process, all possible isomers of TNT are produced; the desired isomer is 2,4,6-TNT (α-TNT). The other isomers are removed from the production product by a sellite process where reaction with sodium sulfite converts the nonsymmetrical isomers to water-soluble sulfonate salts. The 2,4,6-TNT separates from the process water during this reaction (Pearson et al. 1979). Following steam distillation of the process water, distillate water with volatile by-products and impurities including several TNT isomers is discharged to the environment (see Section 7.4.8). Condensate water is recycled or incinerated.

7.4.5.1 Health Effects in Humans

No case reports or epidemiological studies were available.

7.4.5.2 Toxic Effects in Laboratory Animals

Although some sources indicate that the TNT isomers are of the same qualitative and quantitative toxicity (Clayton and Clayton 1982), Zakhari et al. (1978, citing data of Panton and Bates 1921) report that the α compound is less toxic than the β(2,3,4-TNT) isomer and more toxic than the γ(2,4,5-TNT) isomer in cats.

Results of mutagenicity screening of 2,4,6-TNT wastewater condensate products showed that 2,4,5-TNT is the most active compound present in the discharge (Spanggord et al. 1982). It was mutagenic to five strains of Salmonella with or without metabolic activation. However, this compound appears as only a trace component of the discharge (<0.01 ppm). The 2,3,4-, 2,3,6-, 2,4,6-, and 3,4,5-isomers were also tested and found to be mutagenic in one or more strains. In another study the 2,4,5- and 2,3,6-isomers were highly mutagenic to several strains of Salmonella (Dilley et al. 1979). However, of these isomers, only the 2,4,6- isomer is an important component of condensate wastewater.

7.4.5.3 Recommendations and Standards

Industrial hygiene standards have been adopted for only the 2,4,6-isomer (see Section 7.1.3).

7.4.6 Dinitrotoluenes

In the munitions industry dinitrotoluenes (DNTs) are of concern as impurities in TNT, as constituents of TNT condensate wastewaters, and as a coating for propellants. Dinitrotoluenes enter the body by inhalation of the vapors or dust, ingestion, and absorption through the skin (Rosenblatt 1980). Information on human toxicity is available from studies of workers in the munitions industry where DNT was used as a chemical stabilizer in the manufacture of smokeless powder during World War II. Animal toxicity studies concern mainly 2,4-DNT, the main organic component of condensate wastewater.

7.4.6.1 Health Effects in Humans

McGee et al. (1942) studied 154 workers exposed to a dinitrotoluene mixture in which the 2,4-isomer predominated. The chief symptoms of the group were an unpleasant metallic taste, weakness, headache, loss of appetite, and dizziness. Other symptoms included nausea, vomiting, difficulty in sleeping, and pain, numbness, and tingling sensations in the extremities. The main clinical findings were pallor, cyanosis, and anemia. An occasional worker was so sensitive to DNT that signs and symptoms developed within a few hours exposure. No permanent physical impairments were found. In a follow-up study, institution of improved personal and industrial hygiene procedures resulted in a reduction in symptoms and clinical signs (McGee et al. 1947).

Ahrenholz (1980) reported on environmental and medical surveys conducted to evaluate dinitrotoluene and diaminotoluene exposures of workers in a diaminotoluene unit at a chemical manufacturing facility. Dinitrotoluene concentrations ranged from 0.013 to 0.42 m/m^3 during an initial survey and from undetectable to 0.10 mg/m^3 during a follow-up survey. Toluenediamine concentrations ranged from 0.008 to 0.39 mg/m^3 during the initial survey and from undetectable to 0.038 mg/m^3 during follow-up. Medical findings included reduction in sperm count of exposed workers and a slight excess of miscarriages among wives of exposed workers. These findings could not be conclusively related to exposures, however.

7.4.6.2 Toxic Effects in Laboratory Animals

No information on toxicity via inhalation or skin absorption was found. The acute oral toxicities of all tested DNT compounds are generally similar (Table 7.12). In one study 3,5-DNT was the most toxic,

TABLE 7.12. ACUTE ORAL LD_{50} VALUES (mg/kg) OF VARIOUS DINITROTOLUENE (DNT) AND AMINODINITROTOLUENE (ADNT) COMPOUNDS IN RATS AND MICE

Compound	Rats			Mice		
	Males[a]	Females[a]	Both sexes[b]	Males[a]	Females[a]	Both sexes[b]
2,3-DNT	1,102	911	1,122	1,372	1,089	1,072
2,4-DNT	568	650	268	1,954	1,340	1,625
2,5-DNT	616	517	707	652	659	1,231
2,6-DNT	535	795	177	621	807	1,000
3,4-DNT	907	807	177	859	747	1,414
3,5-DNT	309	216		611	607	
2-ADNT	2,240	1,394		1,722	1,522	
4-ADNT	1,360	959		1,342	1,495	

a. Data from Ellis et al. 1978.
b. Data from Lewis and Tatken 1982.

and 2-amino-4,6-DNT (2-ADNT) was the least toxic (Ellis et al. 1978). Acute toxic effects were exerted primarily on the central nervous system: inactivity, unconsciousness, and, in rats given ADNTs or 3,4-DNT, a delayed hyperexcitability and failure to groom. Most deaths occurred within 24 hours of dosing, but delayed deaths in both species and longer lasting effects in rats were observed.

Subchronic (90-day) and chronic (2-year) oral toxicity studies for 2,4-DNT with dogs, rats, and mice were performed by Ellis et al. (1980). Dose levels were reported in terms of (1) the no-effect dose which caused no apparent effects, (2) the toxic dose which caused some adverse effects without death, and (3) the lethal dose which caused an increase in deaths. Doses and effects are listed in Table 7.13. Results for 2,6-DNT were similar. Mice were especially resistant to 2,4-DNT; this is attributable to the low percentage of 2,4-DNT absorbed by this species.

In addition to nonspecific effects of reduced body weight and decreased life span, chronic administration of 2,4-DNT caused effects on a number of target organs in the three species studied, including the blood (methemoglobinemia and sequelae such as Heinz body formation), central nervous system (incoordination, sometimes including ataxia and paralysis), liver (degenerative and hyperplastic lesions, including hepatocellular carcinoma), kidney (cystic changes and tumors), gonads (atrophy and aspermatogenesis in the male, nonfunctional follicles in the female), and skin and mammary gland (fibromas and fibroadenomas, respectively) (Ellis et al. 1979, 1980).

TABLE 7.13. EFFECTS OF ORAL DOSES OF 2,4-DNT IN SUBACUTE AND CHRONIC STUDIES OF DOGS, RATS, AND MICE[a]

	Doses (mg/kg/day)		
	No Effect	Toxic	Lethal
Dogs (gavage)			
13 weeks	5	-	25
2 years	0.2	1.5	10
Rats (in feed)			
13 weeks	0.07%	0.2%	0.7%
	M - 34	M - 93	M - 266
	F - 38	F - 108	F - 145
2 years	0.0015%	0.01%	0.07%
	M - 0.57	M - 3.9	M - 34
	F - 0.71	F - 5.1	F - 45
Mice (in feed)			
13 weeks	0.2%	0.7%	-
	M - 137	M - 413	
	F - 147	F - 468	
2 years	13.5 (0.01%)	95 (0.007%)	900 (0.5%)

a. Data from Ellis et al. 1980.

Of the eight DNT isomers listed in Table 7.12, all were very mild to mild primary irritants to rabbit skin except the 2,5-isomer, which was considered a moderate irritant (Ellis et al. 1978). All were nonirritating to the eyes of rabbits. Only the 2,6-isomer produced mild dermal sensitization in guinea pigs.

In the Ames mutagenicity assay, all DNT isomers were mutagenic in at least one strain of Salmonella typhimurium (Ellis et al. 1978; Pearson et al. 1979) (see Table 7.14). The 2,4- and 2,5-isomers exhibited significant increases in the number of histidine-requiring revertants at 10 or 30 µg per plate. 2,5-DNT showed a positive response in five tester strains. Four isomers, 2,3-DNT, 2,6-DNT, 3,4-DNT, and 3,5-DNT, displayed only weak mutagenic activity (Ellis et al. 1978).

The mutagenicity of technical grade DNT and the six isomers of DNA were determined in three assays with Salmonella typhimurium (Couch et al. 1981). The mixture and the individual isomers were mutagenic, particularly in strains responding to frame shift mutagens. Metabolic activation generally resulted in a decrease in mutagenic response. Neither technical grade DNT nor any of its purified isomers elicited a mutational response when tested on 6-thioguanine resistant Chinese hamster ovary cells, with or without metabolic activation (Abernathy and Couch 1982).

The mutagenic effects of 2,4-DNT were tested in mice using the dominant lethal assay, the sperm morphology test, and the recessive spot test (Soares and Lock 1980). 3,5-DNT was tested in the dominant lethal test. Compounds were administered by both intraperitoneal injection and gavage. Neither of the compounds produced a significant response in any of the test systems.

As previously mentioned, cancers were found in three species of animals subjected to chronic toxicity testing with 2,4-DNT. Rats fed doses of 35 mg/kg/day (males) or 46 mg/kg/day (females) for 2 years had an increased incidence of tumors in various organs (Ellis et al. 1979). Liver hepatomas were evident by the twelfth month. Some hepatic lesions were seen in dogs and mice, but the most severe effect was in rats, where progressive development of hepatocellular carcinomas occurred.

A study by the National Cancer Institute (NCI 1978a) concluded that dietary administration of 2,4-DNT to Fischer 344 rats induced benign tumors (fibroma of the skin and subcutaneous tissue in males and fibroadenoma of the mammary gland in females). No evidence was provided for the carcinogenicity of the compound in B6C3F1 mice of either sex. Ellis et al. (1979) point out that 2,4-DNT is, in part, metabolized to 2,4-diaminotoluene (2,4-DAT). This compound has also been bioassayed by NCI (1978b) and found to be carcinogenic in both rats and mice. In the rats, hepatocellular carcinomas or neoplastic nodules occurred at incidences that were dose related in both the males and the females. In addition, carcinomas or adenomas of the mammary gland occurred in the female rats. In the male rats, fibromas of the subcutaneous tissue occurred. In female mice, NCI found hepatic carcinomas while Ellis et al. (1979), using a different strain of mice, found kidney tumors. In

TABLE 7.14. SYNTHETIC CONDENSATE WASTEWATER MIXTURE[a]

Compound	Concentration (mg/L[b])	Relative Concentration (%)	Ames Mutagenicity[c]
Toluene	0.200	0.590	-
2-Nitrotoluene	0.030	0.089	-
4-Nitrotoluene	0.100	0.295	+
2,3-Dinitrotoluene	0.400	1.180	+
2,4-Dinitrotoluene	14.700	43.377	+
2,5-Dinitrotoluene	0.400	1.180	+
2,6-Dinitrotoluene	7.300	21.541	+
3,4-Dinitrotoluene	0.500	1.457	+
3,5-Dinitrotoluene	0.520	1.534	+
2,3,6-Trinitrotoluene[d]	0.268	0.791	+
2,4,6-Trinitrotoluene	0.400	1.180	+
2-Amino-4-nitrotoluene	0.033	0.091	+
2-Amino-6-nitrotoluene	0.010	0.030	+
3-Amino-4-nitrotoluene[d]	0.0027	0.080	+
2-Amino-4,6-dinitrotoluene	0.020	0.059	+
2-Amino-3,6-dinitrotoluene	0.030	0.089	+
3-Amino-2,4-dinitrotoluene	1.500	4.426	+
3-Amino-2,6-dinitrotoluene	1.200	3.541	+
4-Amino-2,6-dinitrotoluene	0.600	1.770	+
4-Amino-3,5-dinitrotoluene	0.200	0.590	+
5-Amino-2,4-dinitrotoluene[d]	0.700	2.066	+
1,3,5-Trinitrobenzene[d]	0.153	0.451	+
1,3-Dinitrobenzene	4.000	11.803	+
1,5-Dimethyl-2,4-dinitrobenzene	0.390	1.151	+
3,5-Dinitroaniline[d]	0.058	0.171	+
3-Methyl-2-nitrophenol	0.012	0.035	-
5-Methyl-2-nitrophenol	0.032	0.094	-
2,4-Dinitro-5-methylphenol[d]	0.085	0.251	+
3-Nitrobenzonitrile[d]	0.013	0.035	+
4-Nitrobenzonitrile[d]	0.009	0.027	+

a. From Pearson et al. 1979.

b. These are 90th percentile concentrations based on 7 condensate water analyses.

c. Observation of mutagenicity indicated by +; failure to observe mutagenicity by -.

d. These compounds were not represented in 10% of the samples. The values given represent the mean of the observed nonzero values.

the Soares and Lock (1980) study, 2,4-DAT did not induce dominant lethal mutations or morphologically abnormal sperm in mice; however, the International Agency for Research on Cancer (IARC 1978) reported studies in which 2,4-DAT produced reverse mutations in Salmonella under conditions of metabolic activation and was a weak mutagen in Drosophila melanogaster, inducing sex-linked recessive lethals when fed at a concentration of 12.2 mM. 2,5-Diaminotoluene is a teratogenic agent in mice (Shepard 1980).

In one- and three-generation reproductive studies with rats, there were no teratogenic effects on the offspring from any of the matings (Wolkowski-Tyl et al. 1981; Ellis et al. 1979). In the latter study, adverse reproductive effects such as reduced fertility, reduced litter viability, and reduced number of litters were associated with aging and/or the toxicity of 2,4-DNT. In this same study, the subacute and chronic administration of 2,4-DNT was associated with decreased spermatogenesis in male rats and mice and decreased corpora lutea in female mice. Soares and Lock (1980) also found marked reductions in the percentage of fertile matings of 2,4-DNT and 3,5-DNT treated mice.

7.4.6.3 Recommendations and Standards

The recommended threshold limit value for 2,4-DNT is 1.5 mg/m^3 (skin); the STEL is 5.0 mg/m^3 (ACGIH 1983). The Occupational Safety and Health Administration standard is 1.5 mg/m^3 (skin) (OSHA 1982). The OSHA standard does not indicate a particular isomer; presumably it applies to all 2- and 4- isomers (Lewis and Taken 1982) because it is based on ACGIH documentation, which indicates that the commercial grade of DNT is a mixture of ortho-, meta-, and para-isomers.

7.4.7 TAX AND SEX

Information on the metabolism and biological effects of TAX (1-acetylhexahydro-3,5-dinitro-1,3,5-triazine) and SEX (1-acetyloctahydro-3,5,7-trinitro-1,3,5,7-tetrazine), by-products of RDX and HMX manufacture, was not found. The concentration of TAX and SEX in a sample of RDX from the Holston AAP was estimated to be less than 0.5 percent (Cholakis et al. 1980). The concentration of SEX in the effluent wastewater from the plant was 2.5 ppm (Chen et al. 1981).

7.4.8 Wastewater Discharges

Aqueous effluents from plants that manufacture or process TNT are termed "pink water." The color is due to TNT degradation products produced when TNT solutions are exposed to sunlight or ultraviolet radiation. The nature of the TNT degradation products is as yet ill-defined (Dacre and Rosenblatt 1974). The major proportion of the TNT degradation products, the "pink" constituents, are organic-insoluble anions, which have been little studied. More work has been done on identifying the organic-soluble compounds, a large proportion of which are dinitrotoluenes.

7.4.8.1 Load, Assemble, and Pack (LAP) Wastewaters

Untreated wastewaters from Army ammunition plants conducting load, assemble, and pack operations contain TNT and RDX in a ratio of approximately 1.6:1. The toxicity, mutagenicity, and cytogenicity of photolyzed and nonphotolyzed samples of LAP effluents have been studied and results have been compared to results for TNT and RDX alone (Dilley et al. 1978a, 1982b). Photolyzed LAP [hereafter referred to as LAP(I)] was prepared by ultraviolet irradiation of a representative LAP mixture; LAP(I) thus contains the photodegradation products of the LAP chemicals. LAP wastewater was moderately toxic to rats and mice. Acute oral LD_{50}s of LAP wastewater were 947 and 1,131 mg/kg in male and female mice, respectively, and 574 and 594 mg/kg in male and female rats, respectively. In rats, the acute toxicity of LAP wastewater is thus between that of TNT and RDX, whereas in the mouse, it is less toxic than either component (see Tables 7.4 and 7.6). The ultraviolet irradiation of LAP wastewater increased its acute toxicity to mice. Acute oral LD_{50}s were 585 and 684 mg/kg for male and female mice, respectively. In a subacute study with rats, however, the LAP(I) was less toxic than the unirradiated mixture when incorporated into the diet.

All animals given a lethal dose of LAP wastewater died within 24 hours after dosing, after having convulsions of the grand mal type. Animals that survived the convulsions also survived the 14-day post-treatment observation period.

The subacute toxicity of LAP wastewater was studied for 90 days in dogs, rats, and mice (Dilley et al. 1978a). Body weights, weight gain, and food intake were suppressed in all three species. A type of anemia similar to that observed with TNT was produced, with enlarged and hemosiderotic spleens and red urine. In dogs and rats, testicular atrophy, liver enlargement, and serum cholesterol and/or serum triglyceride elevations were observed. Effects on the uterus were observed, the most clear-cut being hypoplasia in rats. Dogs and rats exhibited numerous neurological and clinical signs of toxicity, which were more severe in the dogs. These included convulsions, paresis of the hind legs, inactivity (followed by hyperactivity), ataxia, head-bobbing and/or -swinging, and diarrhea. At the highest dose levels for dogs (50 mg/kg/day), rats (0.5 percent in the diet), and mice (0.5 percent in the diet), mortality was up to 50 percent or more. Most of the observed effects suggest that the toxicity of LAP wastewater is dominated by the TNT component.

In rabbits, LAP and LAP(I) wastewaters were eye irritants [LAP(I) irreversibly] but were mildly or only slightly irritating to the skin (Dilley et al. 1978a). They were classified as strong allergens in the skin sensitization test in guinea pigs.

Both LAP and LAP(I) wastewaters were mutagenic in the Ames mutagenicity assay (Dilley et al. 1978a). LAP(I) wastewater was a more potent mutagen, producing an increase in Salmonella revertants in more strains

than did LAP wastewater and at much lower concentrations (5.0 µg vs 5,000 µg). Because the percentage content of unreacted TNT is much lower in LAP(I) than in LAP wastewaters, the test results suggest that ultraviolet irradiation produces decomposition products that are more potent mutagens than TNT.

In in vivo studies, cytogenetic analyses of bone marrow from rats fed TNT or LAP wastewater showed that neither caused mutations (Dilley et al. 1978a). Since TNT causes microbial mutations in vitro, it was hypothesized that either the rats ingested insufficient quantities of the compound to induce genetic damage or the compounds were metabolically deactivated before reaching the bone marrow.

LAP wastewater gave a positive response in the unscheduled DNA synthesis (UDS) assay in a cell line derived from human fibroblasts, but only in the presence of metabolic activation (Dilley et al. 1978a). This is in contrast to TNT, which gave a positive response only when metabolic activation was not used, and to RDX, which was negative under both conditions.

Using EPA-suggested guidelines, Dilley et al. (1978a) calculated an Acceptable Daily Intake of LAP wastewater which would produce no likely toxic effects in humans. Depending on the reference species used in the subacute toxicity tests, this value ranged from 0.50 to 8.28 µg/kg of body weight. The calculated upper limit range for LAP effluent in water bodies was 16.2 to 268 µg/L (ppb).

7.4.8.2 TNT Condensate Wastewater

Condensate from the distillation of red water is discharged into the aquatic environment. This condensate contains volatile TNT production by-products and impurities (mostly nitroaromatics) that vaporize with the water during the distillation process. The organic constituents have been identified and quantified, and, through the use of statistical procedures, a mixture representative of the actual wastewater has been defined (Pearson et al. 1979). Only 22 of the 30 compounds identified were consistently detected above the ppb detection limit. The major component is 2,4-DNT. The 30 compounds and their relative concentrations are listed in Table 7.14. All 30 compounds were individually subjected to aquatic toxicity tests (Section 8.5.1) and mutagenic screening. Mixtures were subjected to acute oral toxicity tests.

The acute oral LD_{50}s of three condensate water mixtures, differing in the number and relative concentration of the components, ranged from 250 to 450 mg/kg in the rat (Dilley et al. 1979). This is in the range reported for 2,4- and 2,6-DNT, components that constitute 65 to 75 percent of the mixture.

In subacute toxicity studies with dogs, rats, and mice using condensate water mixtures, all three species exhibited a mild compensatory anemia, effects on the brain and central nervous system, and alterations in the liver and spleen (Dilley et al. 1979). In addition, body

weights, weight gains, and food intake were suppressed in mice and rats. Males had testicular atrophy, and females had hyperplasia of the uterus (rats) or inflammation in the tubular reproductive tract (mice).

Most of the compounds in Table 7.14 were positive in the metabolically activated Ames test for mutagenicity (Dilley et al. 1979). The exceptions were toluene, 2-nitrotoluene, 3-methyl-2-nitrophenol, and 5-methyl-2-nitrophenol, all relatively minor constituents.

In rabbits, condensate wastewater was nonirritating to the eyes and only mildly irritating to the skin. It was considered a moderate allergen in the skin sensitization test in guinea pigs (Dilley et al. 1979).

Based on the highest dose levels at which no effects occurred in laboratory animals, an Acceptable Daily Intake range of condensate water for man was estimated to be from 0.50 to 1.16 µg/kg (Dilley et al. 1979). Using these values and a bioconcentration factor for the mixture derived from octanol/water partition coefficients, an upper permissible range for condensate water effluent in water bodies of 15 to 35 µg/L was recommended.

7.4.9 Inorganic Pollutants

In addition to the organic pollutants discussed in Sections 7.4.8.1 and 7.4.8.2, inorganic pollutants are present in wastewater discharges (see Section 8.1.1). Few studies characterize these inorganic pollutants, but two reports (Chen et al. 1981 and Ribaudo et al. 1981) identify heavy metals and inorganic ions present in wastewater effluents from munition plants. Three of the heavy metals (cadmium, chromium, and copper) are of concern because of their known toxic effects to humans.

7.4.9.1 Cadmium

A vast literature on the toxicological actions of cadmium (Cd) is available. Cadmium and other heavy metals are not a problem during explosives manufacture but are present in discharges to the environment; therefore, only environmental sources, such as drinking water, and the oral route of intake will be considered here. The following information on heavy metals was taken from reviews by Doull et al. (1980) and Stokinger (1982b).

Depending on the animal species, the absorption of cadmium from the gastrointestinal tract is relatively minor, ranging from 0.5 to 12 percent. The halogen salts, sulfate, and nitrate are relatively soluble in water, while the oxide, hydroxide, and carbonate are insoluble.

The distribution in the tissues of rats of $CdCl_2$ administered in drinking water (six levels from 0.1 to 50 ppm Cd) was as follows at the highest level: bone, <1 µg/g; kidney, 50 µg/g; and liver, 40 µg/g. Following ingestion of cadmium with carrier by rats, 88 percent of the cadmium was fecally excreted, and only about 1 percent was excreted in the urine. Little or no cadmium was found in the bone or soft tissues. The major route of cadmium excretion in humans is the urine.

After prolonged exposure, about 50 percent of the body burden of cadmium is found in the liver and kidney and the rest is widely distributed in other tissues. Cadmium is highly cumulative. Human autopsy data show that concentrations rise to a peak in the kidney at age 50 in humans.

In experimental animals, the acute lethal dose varies from 100 mg/kg for soluble salts of cadmium to several thousand mg/kg for both metallic cadmium powder and the insoluble salts. Respective estimated lethal doses in humans range from 350 to 8,900 mg. As little as 14.5 mg taken orally has caused nausea and vomiting, but as much as 326 mg was not fatal. In reported accidental ingestion, 13 to 15 ppm cadmium in popsicles has sickened children, as has 67 ppm in punch, and 530 ppm in gelatin.

Cadmium is a powerful emetic; the toxic responses following oral intake include nausea, vomiting, salivation, diarrhea, and abdominal cramps. In acute poisonings where death is delayed for one or two weeks, renal and cardiovascular failure may occur. Extensive damage to the liver may also occur.

In chronic poisonings, the organ effects depend on the route of intake. In inhalation exposures of workers, the lungs appear to be the critical organ, probably because they are the first sensitive tissue encountered in the passage of cadmium into the body. Industrial inhalation exposure results in pulmonary emphysema. Other organ systems, most commonly the urinary system (as evidenced by proteinuria), are also affected.

In contrast, the skeletal system appears to be the target organ in chronic dietary intake. The Japanese itai-itai disease was attributed to high intake of cadmium in people living in certain areas of Japan where water and crops were polluted by industrial cadmium wastes. The most prominent effects were osteomalacia with attendant spontaneous multiple bone fractures, particularly in multiparous postmenopausal women.

In 1965 it was suggested that there is a relationship between cardiovascular disease (hypertension) and cadmium in drinking water supplies (Doull et al. 1980, citing data of Schroeder 1965). This is a controversial issue and has not been fully resolved. Cadmium is also of concern because of its metabolic interrelationships with other environmental trace elements, its effects on the blood, kidney, prostate, and testes, and its possible carcinogenic action. Although several surveys of industrial workers exposed to cadmium fumes show an excess of total malignant neoplasms, particularly prostatic cancer, several studies in which cadmium was administered in drinking water or intragastrically to laboratory animals resulted in no excess tumors. The International Agency for Research on Cancer (IARC 1973) in its review of cadmium as a carcinogen found that the data available at the time were insufficient to reach a conclusion.

The National Interim Primary Drinking Water Regulations, in accordance with the provisions of the Safe Drinking Water Act (Public law 93-523), set a maximum contaminant level for cadmium in community water systems of 0.01 mg/L (Environmental Protection Agency 1976).

7.4.9.2 Chromium

Chromium (Cr) exists in several valance states. Only the trivalent and hexavalent states are biologically significant. Trivalent chromium is an essential element in animals.

Concentrations in water supplies normally range from <10 ppb (natural water supplies) to 35 ppb (municipal drinking water). Absorption of chromium is limited to 1 percent of the daily intake. The major environmental source of chromium is food.

Absorption, distribution, and excretion depend on the valance state, salt form, and route of administration. Distribution studies of chromium in rats fed Cr^{6+} in drinking water at various levels (0.45 to 11 ppm) for one year showed highest amounts in the spleen followed by bone, kidney, and liver. At 25 ppm Cr^{3+} in the water, the amount retained was one-fifth to one-tenth that of Cr^{6+}. Urinary excretion accounts for the elimination of 80 percent of injected chromium; elimination also occurs via the intestine.

Soluble chromates are of very low toxicity by mouth (TDLO of ~1500 mg/kg). Industrial exposure (skin contact and inhalation) to Cr^{6+} compounds has led to a variety of severe toxic effects including dermatitis, penetrating ulcers on the hands and forearms, perforation of the nasal septum, and inflammation of the larynx and liver. Epidemiologic studies indicate that chromate is a carcinogen with bronchogenic carcinoma as the principal lesion.

The occurrence of chromium in food or water has not been shown to produce any significant adverse effects in either humans or laboratory animals. Hexavalent and trivalent chromium proved nontoxic to rats when given in drinking water at levels from 0.45 to 25 ppm. In another study, incorporation of hexavalent chromium (5 ppm) in the drinking water of mice over their lifetimes produced a slightly higher incidence of malignant tumors than in the controls. Trivalent chromium (chromium acetate) given to rats under similar conditions produced no such effect.

The National Interim Primary Drinking Water Regulations' maximum contaminant level for chromium in community water systems is 0.05 mg/L (Environmental Protection Agency 1976).

7.4.9.3 Copper

Copper is an essential trace element in most organisms. It is absorbed primarily in the stomach where the acidic condition favors solubility. Most of the daily intake by humans is excreted in the feces. In the blood, it is bound to two proteins. Excess copper is stored in the liver and bone marrow.

Except for metal fume fever (inhalation route), effects from acute or chronic exposures to copper are rare. Acute poisonings resulting from ingestion of excessive amounts result in vomiting, hematemesis, hypotension, melena, coma, and jaundice. Autopsy findings revealed centrolobular hepatic necrosis and biliary stasis. Copper poisoning in animals leads to injury of the liver, kidney, and spleen. The most toxic copper salts are the sulfate and chloride. The LD_{50} (rat) for $CuCl_2$ is 140 mg/kg. The LD_{LO} for humans for this compound is 200 mg/kg.

One source (Doull et al. 1980) indicates that excessive copper exposure in normal persons does not result in a chronic disease. However, Stokinger (1982b) reviews a symposium on the relationship between Wilson's disease and copper metabolism. In humans, Wilson's disease, typified by chronic copper poisoning, has symptoms of hepatic cirrhosis, brain damage and demyelination, and kidney defects. Excess copper deposition occurs in these affected organs and in the cornea of the eye.

The National Secondary Maximum Contaminant Level for copper in public water systems is 1 mg/L (Environmental Protection Agency 1979).

7.5 CONCLUSIONS

7.5.1 Health Effects

A review of the literature on the toxicity of TNT, RDX, and HMX indicates that these compounds are only moderately acutely toxic. LD_{50} values for the three respective compounds, when administered orally to the rat, were approximately 1000, 100, and >100 mg/kg. Acute exposures with toxic reactions in humans have occurred in the past, primarily when production rates were high during World Wars I and II and when there was less emphasis on industrial hygiene procedures. Health effects such as deaths, hematologic changes, and convulsions have been correlated with exposure levels for TNT and RDX, and standards for industrial exposures have been set. Little information on the health effects of HMX and monitored levels in the workplace exist, but available data do indicate that HMX is less toxic than TNT and RDX. Of the waste constituents and by-products of munition production, tetranitromethane (TNM) and, to a lesser extent, nitrogen oxides have the potential to produce acute toxic effects. TNM is highly toxic via the inhalation route. At TNT production sites, TNM is converted to the less toxic trinitromethane. Workplace standards for TNM and NO_x have been set.

Although the acute exposure potentials are low, the potential for chronic exposures to these compounds exists in the manufacturing environment, where fumes and dusts may be generated, and in areas near production sites, where the compounds may be discharged to local waterways or leach into potable groundwater. In the production of munition compounds, significant amounts of wastewater are generated and discharged to the environment. In addition to munitions compounds, the wastewaters contain organic and inorganic by-products for which there are limited toxicological and environmental fate data. Also very little information on toxicological interactions among these compounds is

available. The possible long-term hazards to the general population of low levels of these munition compounds and their associated by-products are of concern.

Levels of munition compounds and their by-products in wastewater discharges and in receiving water bodies have generally not been associated with observable toxic effects. Of primary concern are the long-term effects on mammalian systems from the ingestion of chemicals present in LAP (load, assemble, and pack) wastewater and condensate wastewater. At low levels, many of these compounds and their metabolites are mutagenic (TNT isomers and metabolites, DNT isomers and metabolites, and TNM) and carcinogenic (2,4-DNT and 2,4-diaminotoluene). When administered chronically, TNT and 2,4-DNT were associated with adverse reproductive effects in rats. The major component of condensate wastewater from TNT manufacture is 2,4-DNT. LAP wastewater contains both TNT and RDX. In toxicity tests of LAP wastewater on mammals, there was no evidence of synergism between TNT, the principal constituent of LAP, and RDX. Studies on other compound interactions using mammalian systems are lacking. Additional research aimed at setting standards for these compounds in drinking water is needed.

7.5.2 Data Gaps and Research Recommendations

Available information from the literature is insufficient to adequately assess the potential health threat to humans from munitions production. Areas in which additional data may be helpful in assessing hazards, both in the workplace and in the environment, from release of wastewater are listed below.

1. TNT: a test for detection of the early, symptomless stages of TNT toxicity in humans; a retrospective epidemiological study of workers involved in TNT production during World War II for carcinogenicity; mechanism of testicular atrophy in rats; toxicity of isomers and of metabolites; acute dermal and inhalation LD_{50} values.

2. RDX: identification of intermediate metabolites; mechanism of toxic action; inhalation LD_{50} values.

3. HMX: tests on all aspects of toxicity; metabolism; epidemiological studies including monitoring levels at production facility.

4. DNTs: more research concerning their mutagenicity and carcinogenicity, including carcinogenicity of metabolites; methods for removal from wastewater.

5. SEX and TAX: presence/concentrations in wastewaters and receiving water bodies; toxicity studies.

6. Additional "no observable effect levels" studies on all munitions compounds using mammalian species in order to set Acceptable Daily Intake standards for humans. (Initial studies have been done for TNT, LAP, and condensate wastewater.)

8. ENVIRONMENTAL ASPECTS OF THE EXPLOSIVES TNT, RDX, HMX, AND THE WASTES ASSOCIATED WITH THEIR MANUFACTURE

8.1 ENVIRONMENTAL ENTRY

8.1.1 Aquatic Discharges

8.1.1.1 TNT Associated

The primary aquatic port of entry for 2,4,6-trinitrotoluene (TNT) is direct waste discharges from the Army Ammunition Plants (AAPs). The degree of waste treatment varies from site to site but generally is of a limited nature. The discharges contain several types of pollutants, including nitrogen species, sulfur, phosphates, munition compounds, and solids. These pollutants are not exclusive to AAP discharges but do significantly affect the environment associated with the AAPs. Data are available for several of the AAPs concerning their discharge characteristics and can be compared to recommended levels or standards.

Effluent discharge data for the Volunteer AAP are given by Sullivan et al. (1977) for the October to December period of 1974. As shown in Table 8.1, the nitrogen species, sulfates, and munitions that were discharged exceeded limitations set by the National Pollutant Discharge Elimination System (NPDES) of the Environmental Protection Agency (EPA). These discharges did result in detectable effects on the associated water bodies (see Section 8.4.1.1). Discharges from the AAPs also contain a variety of inorganic compounds (Table 8.2) that arise during manufacture and purification of TNT (Ribaudo et al. 1981). The heavy metals found in the effluent are thought to originate in the stainless steel reaction vessels and holding tanks and are leached out by the acid mixtures. Many of the discharged inorganic components including copper and chromium fail to comply with the Federal Environmental Pollution Standards and Tennessee State Pollution Standards. Analysis of service water into the plant (copper, 0.01 ppm; chromium, not detected) indicates that these ions enter the discharge water within the plant. Since the Volunteer AAP discharge water is alkaline, most of the elements hydrolyze and precipitate; but these hydrolyzed elements may be chelated by TNT or its degradation products and may be toxic.

Information on the wastewater effluent of the Radford AAP was also available. Luh and Szachta (1978) reported discharge values for areas of the AAP, depending on their manufacturing role (Table 8.3). As compared to EPA standards, the released pollutants far exceeded acceptable levels. Although the data on munition compounds were incomplete at the time of their report, the data on nitrates indicate considerable release of munition-related nitrogen that exceeded the standards by 5 to 3,400 times. The authors did comment that installation of proposed treatment facilities would reduce the waste input to the aquatic system by 85 to 90 percent. Other data on the characterization of waste effluent discharges were reported by Nay et al. (1974). They gave the average characteristics of continuous-flow TNT wastewater at Radford AAP as containing 20 mg/L of alpha-TNT, 200 mg/L of nitrates (as N), 1,000 mg/L of

TABLE 8.1. NPDES[a] DISCHARGE STANDARDS AND TYPICAL EFFLUENT DISCHARGE DATA FROM VOLUNTEER ARMY AMMUNITION PLANT (VAAP)[b]

Parameter	NPDES Discharge Limitations (lb/day) mg/L		VAAP Monitoring Data			
			Quantity (lb/day)		Concentration (mg/L)	
	Daily Average	Daily Maximum	Minimum	Maximum	Minimum	Maximum
NH_3-N	0.1	0.5	6.9	140	0.21	3.5
BOD[c]	(66)	10	45	2,890	1.6	12.0
COD[d]	-	20	297	713	7.1	25.7
Dissolved solids	750	1,000	4,670	36,000	130	1,000
NO_2-NO_3-N	-	10	76	1,050	2.4	22.2
SO_4	-	250	2,020	15,000	40	300
Suspended solids	30	-	159	1,450	2.0	29.0
TNT[e] + Nitrobodies	0.3	0.5	0	104	0	3.2

a. NPDES = National Pollutant Discharge Elimination System, Environmental Protection Agency.

b. Adapted from Sullivan et al. 1977.

c. BOD = biological oxygen demand.

d. COD = chemical oxygen demand.

e. TNT = 2,4,6-trinitrotoluene.

TABLE 8.2. RESULTS OF CHEMICAL ANALYSES OF DISCHARGED WATERS AT THE VOLUNTEER ARMY AMMUNITION PLANT[a]

Chemical (ppm)	4/30/76	8/17/76	8/20/76–8/23/76[b]	9/03/76	9/25/76
Sodium	12.2	138	55.2	32.3	34.9
Potassium	4.2	0.82	0.73	0.43	0.72
Calcium	0.52	47.5	430	69.5	47.5
Zinc	0.04	0.13	0.05	0.20	0.13
Iron	0.27	0.03	0.01	0.03	0.02
Magnesium	1.11	0.93	0.94	0.87	0.79
Copper	0.01	0.22	0.16	0.09	0.05
Manganese	0.02	0.02	0.30	0.18	0.05
Chromium	0.09	0.16	0.10	0.11	0.12
Lead	0.002	0.01	0.007	0.004	0.03
Silicon	16.6	2.3	1.90	3.3	3.4
Phosphorus	0.007	0.10	0.02	0.003	0.08
Nickel	0.12	0.11	0.22	0.13	0.12
Vanadium	0.009	0.17	0.15	0.034	0.08
Bromine	0.06	0.18	0.99	0.06	0.29
Arsenic	0.09	0.07	0.08	0.09	0.03
Barium	0.017	0.05	0.02	0.02	0.06
Cesium	0.27	0.02	0.06	0.01	0.07
Sulfur	47.5	42.7	48.0	43.0	38.8
Nitrogen (total)	>11.0	>10.0	>50.0	>10.0	>10.0
Strontium	ND[c]	0.13	0.12	0.08	0.17
Cerium	ND	ND	ND	ND	ND
Lanthanum	ND	ND	ND	ND	ND
Aluminum	0.075	0.29	0.03	0.06	0.15
Dis solids[d]	367.0	552.0	1197.0	410.0	334.0
Dis solids/ HCl added[e]	485.0	605.0	2071.0	888.0	330.0
pH	7.90	8.18	7.32	7.60	7.75
SO_4^{2-}	63.6	204.0	995.0	151.0	101.0
Cl^-	13.	71.3	187.0	87.9	17.9
NO_2	1.56	ND	ND	6.8	ND
NO_3	21.0	27.0	295.0	4.6	62.0
Phenol	0.04	0.62	0.86	ND	1.95
HCO_3^-	52.5	134.0	28.6	42.0	71.8
Organic N	1.12	0.56	0.70	0.84	1.12

a. Adapted from Ribaudo et al. 1981.

b. Composite of three days of samples.

c. ND = not determined.

d. Dissolved solids.

e. Dissolved solids after the addition of hydrochloric acid (HCl) to the original water.

TABLE 8.3. SUMMARY OF WASTEWATER EFFLUENT DATA AT RADFORD ARMY AMMUNITION PLANT[a]

Parameter[b] (Unit)	C-Line Ditch	C-Line Waste Acid TP[c]	A,B-Line Waste Acid TP	Solventless Propellant	TNT Plant	Neutralization Pond	Solvent Propellant	Solvent Propellant
Average flow rate (MGD)	0.83	8.20	17.4	0.034	4.32	0.1627	1.09	1.14
Nitrates as N (lb/day)		17,418	23,635		540	88.9		
Appropriate EPA standards		5	1,668		108			
Sulfates (lb/day)		19,295	74,675	22	3,960	2,940		
Appropriate EPA standards		84	7,467		792			
COD (lb/day)	785	14,740	6,670	78	1,800	85.1	5,896	2,223
Appropriate EPA standards		17	6,670				1,663	
BOD (lb/day)	235	6,901	1,885				1,838	722
Appropriate EPA standards			1,885				518	
TDS (lb/day)	881	243,880	384,250	8	29,160	5,580	1,092	1,140
Appropriate EPA standards			83,440					
Total solids (lb/day)	998	247,900	388,600		29,160	5,610	1,192	1,387
TNT (lb/day)						104		

a. Adapted from Luh and Szachta 1978.

b. COD = chemical oxygen demand; BOD = biological oxygen demand; TDS = total dissolved solids; TNT = 2,4,6-trinitrotoluene; MGD = million gallons per day.

c. TP = treatment plant.

sulfates, 70 mg/L of chemical oxygen demand (COD), and 2,000 mg/L of solids. These values represent the raw wastewater prior to the final treatment stages of neutralization and sedimentation.

Ghassemi et al. (1976) reviewed data on wastewater effluents from two earlier studies (1967 and 1972) of Joliet AAP. The data taken in 1967 indicated that the four main effluent streams discharged a daily total of 3.2 tons of nitrates, 72 tons of sulfates, 9.5 tons of COD, and 14.5 tons of suspended solids. These high levels were generated at a period of peak TNT production at JAAP. The original report attributed much of the wastewater problem to equipment malfunction, operational errors, and lack of discipline in "housekeeping," resulting in spills to relatively clean cooling water. The 1972 study focused on the TNT manufacturing line 10, red water disposal, tetryl production, and sellite production. The TNT manufacturing line produced wastewater with average concentrations of 44 mg/L of suspended solids, 702 mg/L of dissolved solids, 16.5 mg/L of nitrates, 153 mg/L of sulfates, and 0.8 mg/L of TNT. The associated cooling waters were also found to contain 115 mg/L of sulfates and 33 mg/L of nitrates, indicating some leakage. The production of TNT generated about 1,900 gallons of red water per batch, and the resulting condensate of this was found to contain high amounts of COD, nitrogen-containing compounds, and 20-22 mg/L of TNT. Sellite production contributed high concentrations of sulfite, sulfate, and total solids. At a flow rate of 0.173 mgd, the average values for these pollutants were 1,340, 476, and 2,096 lb/day respectively. The Load, Assembly, and Pack (LAP) area of JAAP was also evaluated for contributions to the wastewater effluent. This pink water is generated by washing of shells and process equipment. On average, the pink water contained 1,401 mg/L of total solids, 1,265 mg/L of dissolved solids, 178 mg/L of TNT, and 145 mg/L of hexahydro-1,3,5-trinitro-1,3,5-triazine (RDX). However, after treatment at a diatomaceous earth/carbon adsorption facility, the respective levels changed to 1,070, 1,072, 3.7, and 19, which were significant reductions for the munitions.

Other data on LAP wastewater were compiled by Spanggord et al. (1978). They sampled the effluents of various AAPs to determine TNT and RDX levels as well as their ratio. At Iowa AAP the TNT levels of post-treatment effluent ranged from <0.05 to 24.3 mg/L with a mean value of 1.32 mg/L and a TNT/RDX ratio of 0.55. At Milan AAP, the TNT levels ranged from <0.05 to 210 mg/L with a mean of 20.0 and a TNT/RDX ratio of 1.02. The much higher values at Milan were due in part to a lack of treatment because the samples were taken at a sump location.

It is obvious that without waste treatment of some kind the effluent discharged from the AAPs contributes large amounts of TNT and munitions-related pollutants into aquatic systems. The types of waste treatments utilized for munitions are discussed in Section 9. However, the type of treatment employed may merely displace the direction of munition waste input from aquatic to atmospheric or terrestrial and create equally deleterious effects in those areas.

8.1.1.2 HMX and RDX Associated

RDX and octahydro-1,3,5,7-tetranitro-1,3,5,7-tetrazocine (HMX) almost always occur together in AAP discharge wastewater. The association of RDX and HMX in wastewaters results from HMX being a contaminant of RDX manufacture and from their sharing a common manufacturing process (Sullivan et al. 1979).

Samples of the effluent wastewater from Holston AAP, the only plant producing RDX or HMX, were taken in May 1979 and reported by Stidham (1979). The four 24-hour samples of sample mass and flow rates taken from the manufacturing-waste effluent lines were used in calculating the averages of compounds delivered to the river. Values were calculated for the two main munition compounds and the two principal by-products, 1-acetylhexahydro-3,5-dinitro-1,3,5-triazine (TAX) and 1-acetyloctahydro-3,5,7-trinitro-1,3,5,7-tetrazocine (SEX). The daily averages were 156 lb of RDX, 45 lb of HMX, 49 lb of TAX, and 33 lb of SEX. The concentrations, in mg/L, ranged from 0.110 to 16.02 for RDX and 0.090 to 3.36 for HMX. Maximum concentrations of 5.24 and 2.03 mg/L were recorded for TAX and SEX, respectively.

As with the discharges from TNT-producing plants, the effluents from Holston AAP contained inorganic pollutants traceable to the reaction and holding vessels (Chen et al. 1981). These pollutants included anions and cations (Table 8.4) as well as the heavy metals, cadmium, copper, and chromium (Table 8.5). These inorganic pollutants are important not only because of their basic toxicity, but also because of the potential for synergistic or additive interactions with organic components.

Data on RDX levels in the effluents from LAP sites in various AAPs were compiled by Spanggord et al. (1978). At the Iowa AAP, LAP water contained 23.8 to 173 mg/L of RDX with a mean of 80.5 mg/L, prior to carbon column treatment. After passing through the column, RDX content dropped to <0.1 to 24.4 mg/L with a mean of 1.53 mg/L. At the Milan AAP, LAP water is sent to a sump and overflows to a stream. RDX concentrations at the stream ranged from 0.1 to 109 mg/L with a mean of 11.9 mg/L. Two other AAPs also use sumps but combine them with waste lagoons. Louisiana and Lone Star AAP data were given for these areas and had RDX concentration ranges of 6 to 189 mg/L and 5 to 82 mg/L, respectively. The reported values for these last three AAPs may be a little low because of production stoppages and dilution in the lagoons.

Kitchens et al. (1978) reported data from Holston AAP for the amounts of RDX and HMX discharged per day at various operating conditions. They estimated that at full mobilization, minimum values for discharged munitions would be 340 lb RDX per day and 123 lb HMX per day. They also reported values for effluents at several internal sampling points and in the river for RDX, HMX, TAX, and SEX. The average values ranged from 0.01 (river) to 5.5 mg/L for RDX, 0.01 (river) to 2.6 mg/L for HMX, 0.004 (river) to 4.8 mg/L for TAX, and 0 (river) to 2.3 mg/L for SEX.

TABLE 8.4. INORGANIC IONS FOUND IN THE WASTEWATERS (WW) OF HOLSTON ARMY AMMUNITION PLANT FROM RDX AND HMX MANUFACTURE[a]

Ions	Concentration (ppm)			
	WW1[b]	WW2[c]	WW3[d]	WW4[d]
Na^+	21.0	20.9	16.5	16.7
NH_4^+	0.5[e]	2.0	0.2[e]	0.2
K^+	2.0	2.5	2.0	1.7
Cl^-	--	--	20.4	11.1
SO_4^{2-}	71.8	72.2	53.4	43.0
NO_3^-				16.6

a. Adapted from Chen et al. 1981.

b. From RDX manufacture.

c. From HMX manufacture.

d. Composite samples from RDX and HMX manufacture.

e. Value is an approximation.

8.1.2 Atmospheric Releases

Pollutants associated with munition production can enter the atmosphere via several pathways. Evaporation from aquatic effluent streams or waste storage lagoons is a possible port of entry into the environment. The vapor pressures of the munitions are given in Section 2.0 and indicate that most of the compounds are not highly volatile. During the various manufacturing stages, certain air pollutants are generated and released to the atmosphere. The amounts released from the individual production stages depend on the munition production rate and the degree, if any, of emission-control processes. Burning of munitions, munition wastes, or waste filter material has also been evaluated for release of atmospheric pollutants. More detailed analysis of atmospheric entry of munition-associated wastes into the environment is given below.

8.1.2.1 TNT Associated

The majority of data on TNT-associated air pollutants deal with releases occurring during manufacture. Ghassemi et al. (1976) discussed the air emissions associated with the Joliet AAP and its munition production. Table 8.6 provides the air pollutant emissions from various studies of JAAP as summarized by Ghassemi et al. (1976). The appropriate state standards are given in Table 8.7. The principal pollutants found at JAAP from the manufacture of TNT are nitrous oxides, particulates, sulfates, and acid mists. Incineration of the red water wastes

TABLE 8.5. CADMIUM, COPPER, AND CHROMIUM FOUND IN HOLSTON ARMY AMMUNITION PLANT WASTEWATERS (WW) FROM RDX AND HMX MANUFACTURE[a]

Ions		Sample (ppb)			
		WW1[b]	WW2[c]	WW3[d]	WW4[d]
Cd	Avg (6)	0.866	0.320	0.210	0.338
	Std dev	0.019	0.023	0.024	0.019
	Rel std dev	2.2%	7.1%	11.2%	5.6%
	Rel error	+2.1%	-	-10%	-5.4%
Cu	Avg (6)	12.9	10.6	8.6	10.7
	Std dev	0.61	0.56	0.30	0.76
	Rel std dev	4.7%	5.2%	3.5%	7.1%
	Rel error	-3.1%	+1%	-	+11%
Cr	Avg (6)	0.019	0.021	0.015	0.017
	Std dev	0.00096	0.00076	0.00088	0.0011
	Rel std dev	5.1%	3.6%	5.9%	6.5%
	Rel error	-	-	-	-

a. Adapted from Chen et al. 1981.

b. From RDX manufacture.

c. From HMX manufacture.

d. Composite samples from RDX and HMX manufacture.

also releases particulates. Thompson and Kats (1978) reported that isomers of nitrotoluene and tetranitromethane were released to the air in reprocessing of acids in TNT manufacturing. Manufacturing processes (primarily TNT but also RDX) at Holston AAP (HAAP) were also evaluated for air pollution releases (Bender et al. 1977a). Table 8.8 gives the emission rates and the appropriate standards. In addition to the pollutants found at JAAP, release of methyl nitrate was detected at HAAP. Bender et al., (1977b) also looked at the air emissions of Volunteer AAP. Figure 8.1 illustrates for VAAP the general decline in yearly averages of ambient air pollutant concentrations that parallels the decline in TNT production. As shown, particulates and nitrogen dioxide concentrations greatly exceeded state standards.

In a large study of AAP-associated air pollutants and their behavior, Carpenter et al. (1978) reported additional emission data for Volunteer AAP (Table 8.9). These data show that the nitrogen and sulfur oxides were released in the greatest amounts. Carpenter et al. (1978) also provided data on the incineration of TNT and TNT wastes. Generally, munition wastes are handled by open air burning. However, little information is currently available regarding combustion products from such techniques. Carpenter et al. (1978) reported that burning of 3.8 tons of TNT produced 213 lb of carbon dioxide, 570 lb of nitrogen

TABLE 8.6. SUMMARY OF AIR POLLUTANT DISCHARGE DATA FROM MANUFACTURING AREAS OF JOLIET AAP[a]

Source	Particulates		Sulfur Dioxide		Nitrogen Oxides (as NO_2)		Acid Mist (as 100% H_2SO_4)	
	Grain SCF (dry)	lb/day	ppm	lb/day	ppm	lb/day	mg/m^3	lb/day
14 Ammonia oxidation plants					2,750	20,500		
					1,676			
						12,875		
Avg.					2,213	16,437		
16 Sulfuric acid concentrators			65	2,700			205	3,090
2 Sellite plants			8	5			2,500	370
12 Red Water incinerators	0.359	3,530			1,650	16,000		
	0.226	2,568			1,810	24,552		
		2,075						
Avg	0.293	2,274			1,730	20,576		
Power house boilers	2.28	67,000		47,500		10,000		
2 Tetryl acid bubble tower					22,000	2,000		
2 Tetryl fume recovery					12,000	2,740	12,000	1,410
12 Batch tetryl lines						4,740[b]		
						2,400		
Avg						3,570		
12 TNT[c] acid recovery						21,000		
					27,580	12,288		
				0.12	33,615	34,320		
					35,000	55,296		
Avg					32,065	30,726		
12 TNT fume recovery					11,400	17,600		
				0.60	7,100	12,600		
					10,000	17,520		
Avg					9,500	15,907		
12 TNT recovery houses						46,633[d]		
						47,580		
						24,820		
Avg						39,678		
17 Nitric acid concentrators						1,530[e]		

a. Adapted from Ghassemi et al. 1976.

b. Sum of emissions from acid bubble tower and fume recovery sections.

c. TNT = 2,4,6-trinitrotoluene.

d. Sum of emissions from acid and fume recovery sections.

e. Estimate based on data from Volunteer AAP.

oxides, 4 lb of hydrocarbons, and 684 lb of soot. To aid in estimating the products from incineration of TNT, they also ran some computer modeling of the pollutants generated when TNT is burned with various ratios of air (Table 8.10). Generally burning with high ratios of air to TNT results in fewer reactive species and fewer exotic species. Incineration of red water is a large problem, and Carpenter et al. (1978) calculated the amounts of red water formed, the amounts of organics contained in the red water, and the emission rates for JAAP, VAAP, and Radford AAP from red water incineration (Table 8.11). As the authors pointed out, characterization of the emissions from red water is incomplete, and, in particular, the identification of nitrobody species needs more work.

TABLE 8.7. EXISTING AIR POLLUTANT EMISSIONS OF JOLIET AAP VS ILLINOIS EMISSION STANDARDS[a]

Source	Major Pollutants	Applicable Illinois Standards	Estimated Present Emission[b]
14 Ammonia oxidation plants	NO_x	5.5 lb (expressed as NO_2) per ton of acid (100 basis) produced (Rule 207, d,2,A)	27 lb (as NO_2) per ton of acid produced
		Visible emissions: 5, opacity (Rule 207,d,2,B)	No visual emission data available
16 Sulfuric acid concentrators	SO_2	2,000 ppm (Rule 204,f,1,A)	65 ppm SO_2
	Acid mist	0.15 lb per ton of acid produced (Rule 204,f,2)	2.26 lbs acid mist per ton of acid produced
2 Sellite plants	SO_2	2,000 ppm (Rule 204,f,1,A)	8 ppm SO_2
	Acid mist	No specific standards	378 lb/day
12 Red water incinerators	Particulates	0.08 grain per SCF of effluent gas corrected to 12% carbon dioxide (Rule 203,e,2)	0.293 grain/SCF (dry)
	NO_x	No specific standards	
South Power Plant	Particulates	0.1 lb per million BTU input	0.5 lb per million BTU[c]
	Sulfur oxides	1.8 lb per million bTU input on or aft May 30, 1975 (Rule 204,C,1,A)	1.4 lb per million BTU[c]
12 Batch tetryl lines	NO_x	10 lb (as NO_x) per ton of nitric acid (100 basis) used (Rule 107,e,2)	45 lb (as NO_2) per ton of nitric acid used[d]
12 Batch TNT lines	NO_2	10 lb (as NO_2) per ton of nitric acid (100 basis) used (Rule 207,e,2)	52 lb (as NO_2) per ton of acid produced[e]
17 Nitric acid concentrators	NO_x	3.0 lb (as NO_2) per ton of acid (100 basis) produced (Rule 207,d,4A)	3.2 lb (as NO_2) per ton of acid produced
Burning grounds (LAP area)	Particulates	Under certain conditions, limited open burning of explosive wastes is allowed; a permit is required for open burning (Rule 505)	1.2 MM lb of particulates per year[f] no similar quantitative estimates are available for discharge of gaseous pollutants
LAP boilers	Particulates	0.1 lb per MM BTU input (Rule 203,g,2)	0.1 lb per MM BTU heat input[c,g]
	SO_2	0.3 lb/MM BTU input for distillate oil, Rule 204,C,2,B); 1.0 lb/MM BTU input for residual oil, Rule 204,c,2,A)	0.3 lb/MM BTU heat input[g] 0.6 lb/MM BTU heat input[h]

a. Adapted from Ghassemi et al. 1976.

b. Where applicable and unless otherwise noted, based on average emission values listed in Table 8.2 and the estimated production rates, process weight rate, etc.

c. Actual emission dependent on type of fuel used; estimates from Uniroyal Inc., as cited in Ghassemi et al. 1976.

d. It is assumed that 0.61307 lb of 62% HNO_3 and 3.11951 lb of 60% HNO_3 are used for the manufacture of 1 lb of tetryl; total tetryl production capacity is assumed to be 21.3 tons/day.

e. It is assumed that 0.45159 lb of 62% HNO_3 and 0.59769 lb of 98% HNO_3 are used for the manufacture of 1 lb of TNT; total TNT production capacity for 12 TNT lines is assumed to be 726 tons/day.

f. Uniroyal estimate.

g. Uniroyal data, calculated fuel analysis (Union Oil) and standard emission factors (Public Health Service Publication).

h. Uniroyal data, calculated specifications and standard emission factors.

TABLE 8.8. CHARACTERISTICS OF THE AIR EMISSION SOURCES AT HOLSTON ARMY AMMUNITION PLANT[a]

Location	Operation	Types and Amounts of Emissions	Standard
Area A building 8	Central coal-fired heating plant (seven boilers)	Heat input, 903 MBTU/hr[b] Sulfur oxides, 8,020 lb/day Particulates, 16,000 lb/day	1.6 lb/MBTU (2-hr max) 0.18 lb/MBTU (daily)
Area B building 200 and 222	Central coal-fired heating plant (six boilers)	Heat input, 1,271 MBTU/hr Sulfur oxides, 11,060 lb/day Particulates, 16,000 lb/day	1.6 lb/MBTU 0.18 lb/MBTU
Area B building 334	Nitric acid concentrators	Nitrogen oxides, 5,220 lb/day	
Area B, buildings 302 and 302B	Ammonia oxidation process units	Nitrogen oxides, 17,000 lb/day	
Area B,	Two open burning area	Particulates 1,410 lb/day	No open burning
Area A, building 10	Producer gas plant	Visible emissions, particulates	20% opacity 5 min/hr (max) or 20 min/day (max)
Area A, buildings 2 and 6	Acetic acid and acid anhydride units	Methyl nitrate, 533 lb/day[c] Solvent vapors, 1,067 lb/day Acid mist, 0.001 lb/hr	 Acid mist, 0.009 lb/hr

a. Adapted from Bender et al. 1977a.

b. MBTU = Thousand British thermal units.

c. Methyl nitrate no longer vented to atmosphere.

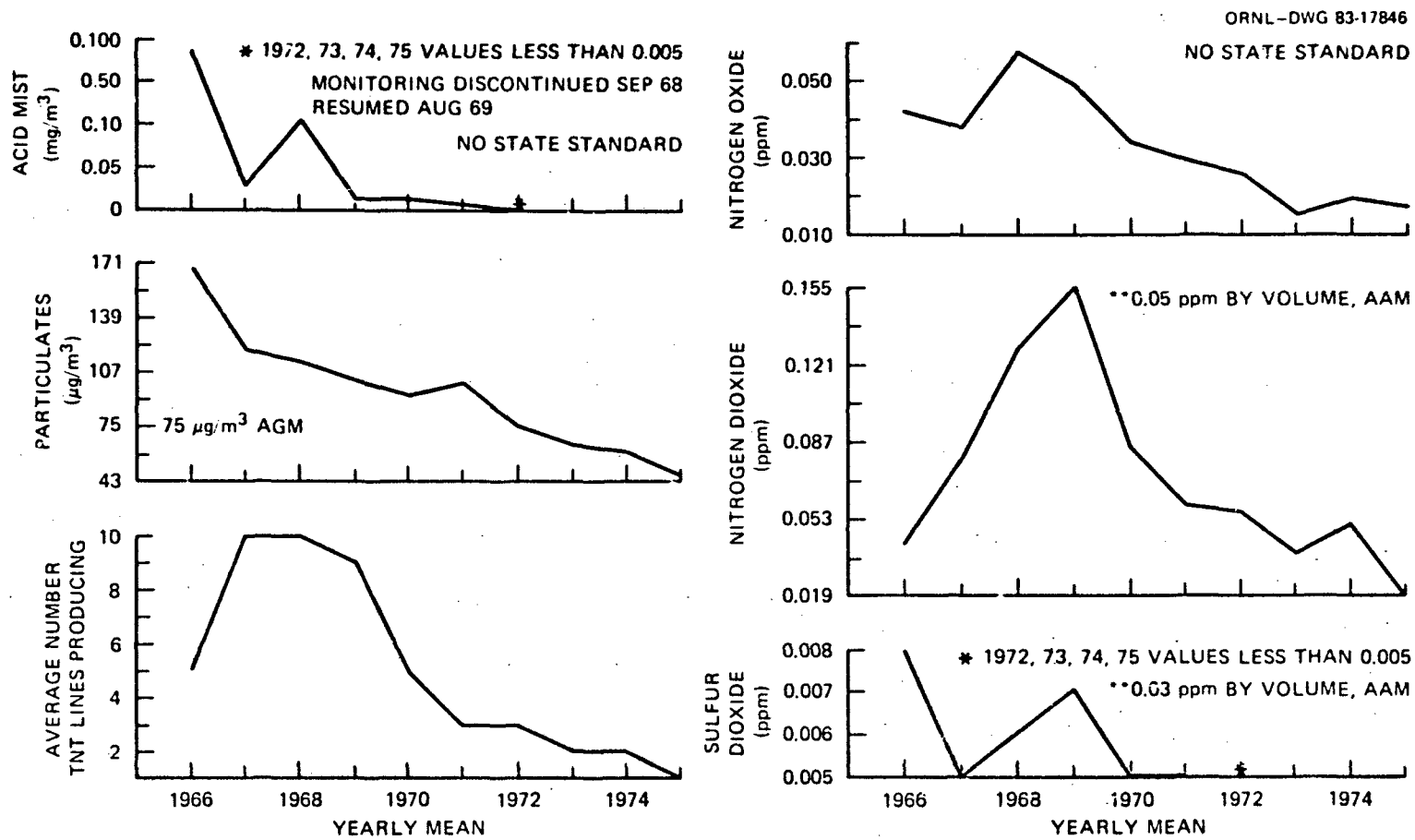

Figure 8.1. Ambient air-quality monitoring data from Volunteer Army Ammunition Plant. From Bender et al. 1977b.

TABLE 8.9. EMISSIONS SUMMARY FROM TNT MANUFACTURING AT VOLUNTEER AAP[a]

Compound	Emission Rate	
	lb/day	ton/year
Group 1. EPA Criteria Pollutants		
Particulates	200	37
Sulfur oxides	2,894	528
Carbon monoxide	75	14
Nitrogen oxides	6,855	1,251
Nonmethane Hydrocarbons	123	22
Group 2. Organics		
Tetranitromethane	c	c
Trinitrobenzene	c	c
Isomers of trinitrotoluene	c	c
Nitrocresols[d]	c	c
Trinitrobenzaldehyde	c	c
Mononitrotoluenes	c	c
Toluene	c	c
Group 3. Miscellaneous		
Sulfuric acid mist	599	109
Nitric acid mist	c	c
Ammonia	e	e

a. Adapted from Carpenter et al. 1978.

b. Assumes plant operations of six lines at 50 tons/day.

c. Emission rates not available although they are believed to be small.

d. Highly toxic.

e. Ammonia emissions are suspected, with data to confirm.

In addition to the information on incineration of munition wastes at the various AAPs, one report was found on assessment of emissions from a prototype fluidized bed incinerator (Carroll et al. 1979). Using a fluidized bed composed of alumina- and nickel-oxide coated particles, slurry solutions (25 percent munitions by weight) were incinerated and the emissions checked for release of TNT, RDX, inorganic cyanides, hydrogen cyanides, nickel carbonyl, nitric oxides, carbon monoxide, and total particulates. The only significant pollutants identified in the releases were particulates and nitrogen oxides. The particulates were composed of nickel and alumina particles and exceeded EPA standards. However, these levels were still lower than those reported for open air incineration techniques. The nitrogen oxides ranged from 200 to 400 ppm and were below EPA standards for fluidized bed incinerators.

TABLE 8.10. INCINERATION PRODUCTS GENERATED FROM BURNING OF TNT AND RDX[a,b]

Pollutant Formed (g/100 g Munition)	TNT/Air Ratio 440 Temperature 1993°C	TNT/Air Ratio 1.27 Temperature 1969°C	TNT/Air Ratio 1.59 Temperature 2164°C	RDX/TNT Ratio 1.53 Temperature 2305°C
CO	73.64[c]	0.463	2.07	52.32
N_2	18.62	66.61	64.62	30.075
C, Graphite	4.57			
H_2	2.12	0.002	0.009	1.525
HCN	0.845			
H	0.019	0.0005	0.003	0.070
C_2H	0.003			
CHO	0.001			0.001
CN	0.0008			
CH_4	0.0007			
CO_2	0.0005	21.87	23.87	7.674
H_2O	0.0005	3.18	3.67	8.082
CH_3	0.0003			
O_2		6.82	4.35	0.004
NO		0.769	0.891	0.024
HO		0.185	0.365	0.158
O		0.050	0.120	0.009
NO_2		0.0007	0.0005	
N				0.0002
HN				0.00002
H_2N				0.00002
H_3N				0.00002

a. Adapted from Carpenter et al, 1978.

b. TNT = 2,4,6-trinitrotoluene; RDX = hexahydro-1,3,5-trinitro-1,3,5-triazine.

c. Values generated from computer model simulations.

TABLE 8.11. RED WATER FORMATION AND INCINERATION DATA AT SEVERAL ARMY AMMUNITION PLANTS (AAP)[a]

Parameter (unit)	Volunteer AAP	Radford AAP	Joliet AAP
Red water formed			
(lb/day)	415,000	104,000	
(lb/yr)	141,200,000	35,300,000	
Red water Organics			
(lb/day)	59,000	15,000	
	Incineration	Pollutants	
Nitrogen oxides			
(lb/hr)		52.6	
(lb/day)			16,800
Particulates			
(lb/hr)		0.2	
(lb/day)			3,600
Sulfur dioxide			
(lb/hr)		2.7	
(lb/day)			1,150

a. Adapted from Carpenter et al. 1978.

8.1.2.2 RDX and HMX Associated

The air pollutants released from RDX/HMX manufacture and incineration include many of the same products identified for TNT. The principal emissions are nitrogen oxides, sulfur oxides, particulates, and acid mists. Additional pollutants may include nitromethane and methyl nitrate, which are formed during the cleavage of hexamethylene tetramine during RDX manufacture (Thompson and Kats 1978).

Carpenter et al. (1978) identified and quantified (based on maximum AAP operating capacity) many air pollutants from the RDX manufacturing process at Holston AAP (Table 8.12). These include a large group of organics at fairly high emission rates. The pollutants emitted in the greatest amounts included nitrogen oxides, sulfur oxides, particulates, and carbon dioxide. In comparisons with EPA standards for ambient air quality (see Section 8.4.2), the nitrogen oxides concentration at boundary locations exceeds acceptable levels by a factor of 55. This represents significant input into the surrounding atmosphere from RDX manufacture. Data on air pollutants released from burning of RDX-containing explosives and wastes were generated by Carpenter et al. (1978) using computer modeling of Compound B incineration. Compound B is a mixture of 60 percent RDX and 40 percent TNT, and the pollutants resulting from incineration in the absence of air are given in Table 8.10. The pattern of reactive pollutants released is very similar to those generated when TNT is burned at a low air concentration, but more

TABLE 8.12. EMISSIONS SURVEY FROM RDX MANUFACTURING AT HOLSTON AAP[a]

	Compound	Emission Rate[b] lb/day	ton/year
Group I.	EPA Criteria Pollutants		
	Particulates	14,749	2,692
	Sulfur oxides	16,989	3,097
	Carbon monoxide	12,197	2,226
	Nitrogen oxides[c]	64,526	11,776
	Nonmethane hydrocarbons	2,908	531
Group II	Organics	Total (21,745)	(3,969)
	Acetic acid	12,497	2,281
	Acetic anhydride	230	42
	Formic acid	141	26
	Isobutyl acetate	2	0.4
	n-Propyl acetate	1,134	207
	n-Propyl formate	320	58
	Methyl acetate	733	134
	Cyclohexanone	2,278	416
	Acetone	2,754	503
	Methyl ethyl ketone	6	1
	Methyl nitrate	1,558	284
	Nitromethane	30	5
	Methyl- and dimethylamine	18	3
	Toluene	52	9
	Phenol	0.8	0.1
	Trace organics (butanol, propanol, methanol, methyl formate, formaldehyde)	10	2
Group III.	Miscellaneous species		
	Methane	1,955	357
	Hydrogen	418	76
	Carbon dioxide	2,250,000	410,625
	Ammonia	390	71
	Nitric acid	4,409	805
	Explosives (particulates)	2.3	0.4

a. Adapted from Carpenter et al. 1978.

b. Assumes full mobilization as defined in Volume 2, Section 6.A.1, Carpenter et al., 1978.

c. Calculated as NO_2.

CO_2, H_2O, NO, and HO^- are generated from the RDX mixture. Carroll et al. (1979) also tested Compound B in their evaluation of a fluidized bed incinerator. Emissions from burning 5 to 25 percent mixtures of Compound B contained 173 to 388 ppm nitrogen oxides and large amounts of fluidized bed-component particulates. No RDX was found in the gases, but on particulates 0.0004 to 0.0052 kg/hr was detected. These levels were not considered significant and were below guideline values.

8.1.3 Terrestrial Releases and Dumping

Ports of entry for munition compounds to the terrestrial environment are usually indirect, although intentional dumping has been documented. Spills during manufacture, transportation, and storage are one route. The settling on surface soils of airborne particles from manufacturing and incineration is another. Incomplete incineration and dumping of the explosive material in a landfill is also a route of entry associated with a disposal method. Seepage from wastewater ditches and lagoons into the underlying soil structure and associated groundwater is a major route of entry. The primary intentional route of entry is dumping in sanitary landfills.

8.1.3.1 TNT Associated

Data on levels of TNT munitions entering soils are not readily available in the literature. Most data concern levels detected in soils or groundwater after entry. These data are given in Section 8.4.3 on soil and groundwater habitat quality. The remaining information documents the existence of various routes of entry.

Jerger et al. (1976), in an environmental analysis of Iowa AAP, found an abandoned waste lagoon site that after 20 years of disuse had become terrestrial in nature. The lagoon had been allowed to naturally fill in, with the addition of flyash and solid coal wastes from the onsite coal-fired generators. Levels of munition-associated wastes in the soil of this area were found as follows (mg/kg): 0.5 of 2,6-dinitrotoluene (2,6-DNT), 3.0 of 2,4-dinitrotoluene (2,4-DNT), 0.6 of 1,3,5-trinitrobenzene (1,3,5-TNB), 3,030 of 2,4,6-TNT, 101 of 4-hydroxylamino-2,6-dinitrotoluene, and 180 of 2-hydroxylamino-4,6-dinitrotoluene. Pereira et al. (1979) documented the contamination of groundwater below waste disposal beds at Hawthorne Naval Ammunition Depot by TNT at levels up to 620 µg/L.

Bender et al. (1977b) determined that the production area of the Volunteer AAP has been contaminated by spills from transportation and manufacture of TNT. This includes the batch TNT lines, burning grounds, and sanitary landfill. Also, fallout of particulates downwind from the production area was identified as a contamination route. Similar documentation of entry i to soil and groundwater from production lines, disposal sites, and emission fallout is given for Milan AAP by Envirodyne Engineers Inc. (1980), and for Alabama AAP by Keirn et al. (1980). Levels for these areas are given in Section 8.4.3.

8.1.3.2 RDX and HMX Associated

Similar routes of entry are expected for RDX and HMX munitions and wastes as for TNT. The data on TNT contamination at Milan AAP generated by Envirodyne Engineers, Inc. (1980), are accompanied by some data on RDX contamination. Bender et al. (1977a) found contamination of manufacturing areas and disposal sites at Holston AAP which included occasional disposal of sewage treatment sludge and explosive-generated incineration ashes in landfills. This disposal was particularly hazardous because the landfill area was affected by sinkholes and subsidence, indicating that contamination of the groundwater is a distinct possibility.

8.2 ENVIRONMENTAL TRANSPORT AND DISTRIBUTION

8.2.1 Surface Waters

8.2.1.1 TNT Associated

Information on the transportation of TNT and associated compounds within the surface water systems can be obtained from two types of literature reports. Several studies have been conducted in the laboratory or under controlled field conditions to generate data for analyzing how munition compounds move through surface waters. These reports may contain computer modeling or other theoretical estimates. Limited data can also be gleaned from environmental surveys of the various AAPs concerning the concentrations of munitions in the water column and sediments. However, there is a scarcity of data taken far enough downstream to clearly indicate the patterns of munition transportation and distribution.

The best environmental survey of stream transportation of wastes generated from an AAP was performed on Waconda Bay, downstream from Volunteer AAP (VAAP), by Sullivan et al. (1977). Table 8.13 demonstrates the decline in munition concentrations in the water column with distance from VAAP. The concentrations of the majority of the munitions are reduced to the limits of detection in the water by 0.4 to 1.0 mile. Similarly, the concentrations in sediments also decline after 1.0 mile.

A similar pattern of decline in munition concentrations with distance was found at Joliet AAP by Stilwell et al. (1976). They found decreases in the water column and sediment levels of TNT and dinitrotoluenes after 1 to 2 km of stream length (Table 8.14). The pattern shown by these data is not conclusive due to confounding factors. One such factor was the mixing of a wastewater ditch effluent with a relatively nonpolluted stream.

Data for the Iowa AAP indicated a similar pattern of declining of munition waste concentrations in the water column and sediments with distance, but again with some interfering factors (Sanocki et al. 1976). Table 8.15 indicates the decline in concentrations with distances of approximately 1 km or greater, but the effect is not as clear as the

TABLE 8.13. SUMMARY OF MUNITION DATA IN WACONDA BAY AT VOLUNTEER ARMY AMMUNITION PLANT (VAAP)[a]

Distance from VAAP (miles)	TNT[b] Range (Mean)	2,6-DNT[c] Range (Mean)	2,4-DNT[d] Range (Mean)
	Water Column (ppb)		
0.0	25-37 (31)	45 (45)	40-68 (54)
0.4	7-23 (15)	5-80 (32)	16-66 (46)
0.7	<2-3 (2)	14-48 (30)	<2-27 (17)
1.0	0-3 (2)	0-2 (1.5)	0-25 (8)
1.5	0-4 (2)	0-<2 (1)	0-<2 (1.5)
	Sediments (mg/kg)		
0.0	2.4-3.1 (2.8)	NG[e]	NG
0.4	<0.1-0.5 (0.28)	NG	NG
0.7	<0.1-0.32 (0.21)	NG	NG
1.0	<0.1	NG	NG
1.5	<0.1	NG	NG

a. Adapted from Sullivan et al. 1977.

b. TNT = 2,4,6-trinitrotoluene.

c. 2,6-DNT = 2,6-dinitrotoluene.

d. 2,4-DNT = 2,4-dinitrotoluene.

e. NG = not given.

above examples due to several different point sources of munitions waste input to the stream. These inputs occur above site 2, at site 4, above site 5, and above site 7. Generally, site 8, which is 24 km below a input source, parallels the values for both sediment and water column levels found at the control site 1.

In an analysis of the surface waters of the Alabama AAP (AAAP), Keirn et al. (1981) concluded that there was no evidence for migration of munition wastes at or above detectable limits. The associated sediments in these surface water systems contained detectable levels of munitions with higher concentrations in the upper reaches of streams than at lower reaches.

Spanggord et al. (1980) evaluated several munition compounds for environmental fate (loss and movement) in aquatic systems. They utilized laboratory studies of individual processes (e.g., sediment sorption) and computer models of specific AAP situations to generate comprehensive analyses of environmental fates of TNT and 2,4-DNT. Most of their results will be discussed in Section 8.3, but two areas are pertinent to this transportation discussion. They found that sorption

TABLE 8.14. SUMMARY OF MUNITION DATA IN GRANT CREEK AT JOLIET ARMY AMMUNITION PLANT (JAAP)[a]

Sample Site[b]	Distance[c] (km)	TNT[d] Range (Mean)	2,6-DNT[e] Range (Mean)	2,4-DNT[f] Range (Mean)
		Water Column (ppb)		
Red water treatment outflow	0.0	4.2-146 (75)	2.9-167 (85)	7.2-510 (258)
Red water ditch outflow	0.8	25-133 (55)	12-63 (24)	12-21 (16)
Grant Creek below ditch	0.9	7.6-64 (25)	4.6-50 (28)	2.2-44 (12)
Grant Creek at JAAP boundary	1.8	4.0-12 (9)	13-14 (13)	6.0-16 (10)
		Sediment (ppb)		
Grant Creek below ditch	0.9	10-165 (68)	32-426 (176)	59-633 (278)
Grant Creek at JAAP boundary	1.8	43-62 (50)	78-91 (83)	120-167 (142)

a. Adapted from Stilwell et al. 1976.

b. Samples were taken from the red water outfall, the ditch running from the outfall to Grant Creek, and at several sites in Grant Creek in Spring, 1975.

c. Distances are approximate and are calculated from the red water outfall.

d. TNT = 2,4,6-trinitrotoluene.

e. 2,6-DNT = 2,6-dinitrotoluene.

f. 2,4-DNT = 2,4-dinitrotoluene.

of both munition compounds with sediments was only of minor importance in their total fate. Similarly volatilization of the compounds from aquatic to atmospheric environments was a minor component. Thus they concluded that the transportation and distribution of TNT and 2,4-DNT released in aquatic systems were controlled more by flow rates and transformation processes than by partitioning into other media. The low importance of sediment sorption demonstrated by laboratory analyses is generally supported by circumstantial evidence found in surveys of AAP waters. The higher levels in the sediments than in the water column associated with the AAPs may be more an indication of long-term increases than of significant short-term depositions. Their general conclusion on transportation of TNT and 2,4-DNT was that greatest distance would be traversed under cloudy skies in winter at high flows. These are the worst conditions for photolysis and biodegradation, the two primary factors controlling transportation and distribution. Smith et al. (1981) provided some confirmation of the lack of volatilization of 2,4-DNT and 1,3-dinitrobenzene (another munition-associated compound). They tested both these compounds and found that they were controlled by gas phase mass transfer resistance and classified them as having low volatilization potential.

TABLE 8.15. SUMMARY OF MUNITION DATA IN BRUSH CREEK AT IOWA ARMY AMMUNITION PLANT (IAAP)[a]

Sample Site[b]	Distance[c] (km)	TNT[d] Range (Mean)	2,6-DNT[e] Range (Mean)	2,4-DNT[f] Range (Mean)	4-OH[g] Range (Mean)	2-OH[h] Range (Mean)
		Water Column (ug/kg)[i]				
Brush Creek1	0.0	<0.2 (<0.2)	<0.1 (<0.1)	<0.1-<0.4 (<0.2)	<5 (<5)	<10 (<10)
Brush Creek2	3.2	<0.2 (<0.2)	<0.1 (<0.1)	<0.1 (<0.1)	<5-8 (6)	<10 (<10)
Brush Creek3	4.1	<0.2 (<0.2)	<0.1 (<0.1)	<0.1 (<0.1)	<5 (<5)	<10 (<10)
Brush Creek4	5.7	<0.2-8.4 (2.5)	<0.1 (<0.1)	<0.1 (<0.1)	<5-12 (6)	<10-16 (11)
Brush Creek5	8.1	<0.2-6.7 (3.4)	<0.1 (<0.1)	<0.1-0.1 (0.1)	<5-25 (10)	<10-49 (18)
Brush Creek6	13.2	<0.2-0.6 (0.3)	<0.1 (<0.1)	<0.1 (<0.1)	<5-16 (7)	<10-18 (12)
Brush Creek7	13.8	<0.2-15.3 (4.1)	<0.1 (<0.1)	<0.1 (<0.1)	<5-11 (8)	<10-40 (21)
Brush Creek8	24.2	<0.2-5 (1.3)	<0.1 (<0.1)	<0.1-0.1 (0.1)	<5 (<5)	<10-18 (12)
		Sediments (mg/kg)				
Brush Creek1	0.0	(<1.0)	(<0.1)	(<0.4)	(<5)	(<30)
Brush Creek2	3.2	(9.0)	(2.4)	(<0.1)	(8)	(90)
Brush Creek3	4.1	(<0.2)	(<0.1)	(<0.1)	(<5)	(<30)
Brush Creek4	5.7	(18.7)	(<0.1)	(0.1)	(9)	(45)
Brush Creek5	8.1	(0.3)	(<0.1)	(<0.1)	(<5)	(<30)
Brush Creek6	13.2	(1.0)	(<0.1)	(0.1)	(<5)	(<30)
Brush Creek7	13.8	(2.7)	(0.1)	(0.2)	(<5)	(<30)
Brush Creek8	24.2	(<0.2)	(<0.1)	(<0.1)	(<5)	(<30)

a. Adapted from Sanocki et al. 1976.

b. Brush Creek1 = upstream Control; Brush Creek2 = downstream from munition effluent; Brush Creek3 = downstream from process effluent; Brush Creek4 = downstream from historic munition dump; Brush Creek5 = downstream from munition effluent; Brush Creek6 = upstream of sewage treatment plant; Brush Creek7 = downstream from sewage treatment plant; Brush Creek8 = downstream boundary of IAAP.

c. Approximate distance from upper most station; sources of munition input occur between stations 1 and 2, at station 4, between stations 4 and 5, and between stations 6 and 7.

d. TNT = 2,4,6-trinitrotoluene.

e. 2,6-DNT = 2,6-dinitrotoluene.

f. 2,4-DNT = 2,4-dinitrotoluene.

g. 4-OH = 4-hydroxylamino-2,6-dinitrotoluene.

h. 2-OH = 2-hydroxylamino-4,6-dinitrotoluene.

i. Samples were collected in June 1975.

The solubility of munition compounds in water is an important factor in their transportation in surface waters. The values reported for munition compounds generally indicate that they are not extremely soluble (Table 8.16). Because most munition compounds enter the surface aquatic systems in solution, the impact of the low solubility lies mostly with the slow release of adsorbed compounds from associated sediments. This applies to RDX and HMX as well as TNT.

8.2.1.2 HMX and RDX Associated

Information on the transportation of RDX and HMX in surface waters was limited to some reports of levels downstream from Holston AAP and a study by Spanggord et al. (1980) on environmental fate. Stidham et al. (1979) reported levels of RDX and HMX of 79 and 67 µg/L, respectively, at one mile downstream of the last plant effluent. They attributed these high levels to incomplete mixing of effluents and river water. Sullivan et al. (1979) reviewed data from an earlier study by Sullivan

TABLE 8.16. WATER SOLUBILITY DATA

Compound[a]	Solubility (ppm)	Reference
TNT	123	Rosenblatt and Small 1981[b]
TNT	83.4-86.7[c]	Hale et al. 1979
TNT	130	Hale et al. 1979[d]
RDX	18.5-23.6[c]	Hale et al. 1979
2,4-DNT	273	Rosenblatt and Small 1981[b]
TNB	32	Rosenblatt and Small 1981[b]
1,3-DNB	370	Rosenblatt and Small 1981[b]
HMX	2.63[e]	Spanggord et al. 1982

a. TNT = 2,4,6-trinitrotoluene; RDX = hexahydro-1,3,5-trinitro-1,3,5-triazine; 2,4-DNT = 2,4-dinitrotoluene; TNB = trinitrobenzene; 1,3-DNB = 1,3-dinitrobenzene; HMX = 1,3,5,7-tetranitro-1,3,5,7-tetracyclooctane.

b. Citing Spanggord et al. 1980.

c. At 21°C determined by gravimetric, ultraviolet, and high-pressure liquid chromatography techniques.

d. Citing Urbanskii 1964.

e. At 20°C.

et al. (1977) that reported levels of RDX ranging from 0.7 mg/L immediately below the outfall to <0.005-0.07 mg/L at one mile downstream. Wentsel et al. (1979, citing data of Sikka et al. 1978), discussed studies concerning levels in sediments and adsorption coefficients. Sikka et al. determined that RDX had steady state levels of 30 to 40 ppm in clay and organic sediments (in laboratory evaluations), despite an extremely low adsorption potential indicated by partition coefficients.

Spanggord et al. (1980) studied the movements and loss of RDX in the environment in the same manner as for TNT and 2,4-DNT. Again they found little sorption potential for RDX by sediments. However, unlike TNT, RDX did not degrade quickly in aquatic systems, and the major environmental fate was dilution. This indicates that RDX would remain and be transported in surface water for a considerable time and distance.

Along the same lines, Spanggord et al. (1982) tested HMX and found that physical transport processes of volatilization, sediment sorption, and biosorption were not major factors in its environmental transport. They estimated a volatilization rate constant of 2.4 to 7.2 x 10^{-4} day^{-1} ($t_{1/2}$ = 3000 to 1000 days). Thus HMX should be similar to RDX and be transported in surface waters for a considerable time and distance.

8.2.2 Atmospheric Transportation

Information on the transportation of munition-specific air pollutants was not available in the literature. Carpenter et al. (1978) discussed the atmospheric fate of pollutants associated from production and incineration of TNT and RDX, but provided information only on derived pollutants (e.g., nitrogen oxides). Values at the boundaries of Holston AAP for these derived air pollutants were calculated using a computer model (see Table 8.54) that gave some indication of transportation concentrations within a local area. Data on half-lives of these pollutants were also generated by Carpenter et al. (1978) and indicated potential for long-range transportation because of considerable persistence of the pollutants (see Table 8.21). In addition to atmospheric transportation of pollutants or munitions released directly from AAPs, some potential atmospheric transportation exists from volatilization of munitions dissolved in surface waters. However, Spanggord et al. (1978) concluded that TNT, RDX, and 2,4-DNT underwent little volatilization, and it was not a significant transportation factor. This conclusion was further supported for 2,4-DNT and 1,3-trinitrobenzene by Smith et al. (1981).

8.2.3 Soil and Groundwater Transportation

8.2.3.1 TNT Associated

Laboratory studies of the transportation of TNT-associated munitions through soils and in groundwater have been performed by several researchers. Conley and Mikucki (1976) used cylinders (10 ft high by 5 ft in diameter) filled with soil and/or refuse to evaluate the migration of a 25 mg/L dose of TNT. Leachate was collected for 22 months, and soil samples were analyzed at the end of this period. Virtually zero migration of TNT into the leachate occurred, with levels ranging from 0 to 35 ppb in monthly samples. Soil samples indicated only a small potential for migration of TNT. Osmon and Andrews (1978) also used laboratory cylinders to simulate soil systems. Various soil mixtures of 7-inch depth were tested for TNT migration over a 50-week period. In general they found that TNT moved fastest through clay (probably due to microchannels) and slowest through loam soil. Field studies were then undertaken to evaluate migration under outdoor conditions. A 0.5 kg sample of TNT was placed in an 8-inch-deep hole, and after 40 weeks no more than 3 inches of migration was found. Another 6-month laboratory study of soil migration utilized 5-ft-deep by 3-ft-wide cylinders with sampling ports at 12-to-30-inch intervals (Hale et al. 1979). This study was supplemented by ^{14}C-labeled TNT studies in 24-inch-deep columns. In the main study, little migration of TNT was found, with most TNT levels in water being less than 0.5 ppm. Soil samples confirmed the lack of migration of TNT, with levels dropping off rapidly with increasing soil depth. The treatment level of approximately 20,000 ppm in the upper 10 cm dropped to concentrations of thousands and hundreds of ppm in the next two lower 10-cm sections of soil. Below the 60-cm depth, concentrations remained below 300 ppm. The concentrations varied depending on the soil composition. The ^{14}C TNT soil studies

indicated slightly more migration in water, particularly in the coarse-textured, sandy-loam soil column. However, the concentrations in soil and leachate water after 20 weeks remained below 0.5 ppm. Kaplan and Kaplan (1982) found similar results with 25-cm-high columns of high porosity soils containing 50 ppm (600 mg/column) of TNT. They detected TNT concentrations in water leachates of up to 15 ppm during the first 35 stabilization days and down to 1-2 ppm after 95 days. Thus, even a worst case scenario produced only small amounts of TNT migration in laboratory studies.

Field surveys have shown some migration of TNT in soils and groundwater. Pereira et al. (1979) reported the detection of up to 620 µg/L of TNT in groundwater downgradient from a disposal landfill. Envirodyne Engineers, Inc. (1980), found levels of TNT and 2,4-DNT in groundwater and soils from Milan AAP. Groundwater from depths of 204 to 3,609 cm were sampled and contained TNT levels of <10 to 3,000 µg/L and <10 to 70 µg/L of 2,4-DNT. Associated soil samples from the upper 6 inches contained <2 to 230 ng/g of TNT. This indicates the potential for long-term soil migration of TNT munitions. Alabama AAP was also sampled for contamination of soils and groundwater (Keirn et al. 1981). Keirn et al. found two wells (with depths no greater than 50 ft and at least 15 ft deep) with TNT values of 75 and 10,270 ppb. These sites had corresponding soil contamination of up to 3,000,000 ppb TNT. However, other sites within AAAP with soil contamination (up to 900 ppb at 1 meter of depth) did not display groundwater contamination. In discussing the potential for migration in soils and groundwater of TNT at AAAP, Keirn et al. calculated maximum rates of 2 cm per year for horizontal migration and 25 cm per year for vertical recharge to the aquifer.

8.2.3.2 RDX and HMX Associated

Data for migration of RDX and HMX in soils were found in the laboratory study by Hale et al. (1979) and in the field surveys of Milan AAP by Envirodyne Engineers, Inc. (1980). The laboratory studies were identical to those described for TNT, and a similar degree of migration for RDX was found. Levels of RDX in water leachates were less than 0.5 ppm in both the main study and the ^{14}C study. The soil samples showed a pattern similar to that shown by TNT, with decreases in concentration from levels of approximately 20,000 ppm in the upper 10 cm to 150 ppm at 60 to 90 cm of depth. Again soil types affected the specific values, with coarse loamy soils showing the most migration. Groundwater samples taken at MAAP showed RDX levels of <20 to 780 µg/L, with corresponding soil levels of 51 to 83,000 ng/g (Envirodyne Engineers, Inc. 1980). Thus, RDX and presumably HMX have the same potential for migration in soils and groundwaters as does TNT.

The low solubilities of munition compounds in water (Table 8.16) have a significant effect on groundwater transportation. Since solubilities are low the migration of munitions through soils is slow and only at low concentrations in groundwater (Hale et al. 1979). The partition coefficients between sediments and water also indicate that the munitions will adsorb on sediments (Table 8.17) and be slowly released to water. This applies to TNT as well as RDX.

TABLE 8.17. PARTITION COEFFICIENT EXPERIMENT DATA[a]

Soil[b]	Concentrations (ppm)			p[3]
	Initial Aqueous	C_w[c]	C_s[d]	
		TNT[f]		
Brookston (sicl)	28.6	24.3	122.6	5.0 (14.4)[g]
Genesee (sil)	28.6	23.6	117.6	4.9 (17.8)
Princeton (sl)	28.6	25.7	64.6	2.5 (8.2)
Bennington (sil, acid)	28.6	24.5	31.0	3.3 (13.5)
		RDX[f]		
Brookston (sicl)	23.5	28.0	219.3	7.8
Genesee (sil)	23.5	31.6	200.8	6.4
Princeton (sl)	23.5	22.6	4.8	0.2
Bennington (sil, acid)	23.5	29.1	52.8	1.8

a. Adapted from Hale et al. 1979.

b. Brookston = fine textured silty clay loam soil; Genesee = alkaline, medium-textured silt loam soil; Princeton = coarse-textured, sandy loam soil; Bennington = acidic, medium-texture silt loam soil.

c. C_w = concentration in the aqueous phase.

d. C_s = concentration in the soil phase.

e. p = ratio between solute in soil/water phases.

f. TNT = 2,4,6-trinitrotoluene; RDX = hexahydro-1,3,5-trinitro-1,3,5-triazine.

g. Values in parentheses are calculated using the concentration difference in the water phase to determine C_s.

8.3 PERSISTENCE, BIOACCUMULATION, AND ENVIRONMENTAL DEGRADATION

Persistence of munition compounds in the environment is dependent on several factors including the degree to which they bioaccumulate and the processes that degrade them. This section will first discuss the overall question of persistence and then examine in more detail some of the individual factors involved.

8.3.1 Persistence

The persistence of munition compounds in the environment is dependent on the various mechanisms available to degrade or utilize the compounds and the rate at which these mechanisms can operate under normal environmental conditions.

8.3.1.1 Persistence of TNT and Associated Wastes

In the aquatic environment, mechanisms affecting persistence include the physical degradation processes of photolysis and hydrolysis as well as biological degradation and utilization by bacteria. A laboratory study of the impact of photolysis and microbial degradation on TNT and 2,4-DNT was reported by Spanggord et al. 1980. Both processes were important regarding the persistence of TNT, but in natural waters biotransformation is considerably slower (under certain conditions as much as 1,000 times slower) than photolysis. The photolysis half-lives of TNT vary with season and latitude, as shown in Table 8.18. Studies of microbial degradation indicate that the half-lives are longer (19 to 25 days) and also occur after a lag period of 13 to 20 days. The rate of transformation is increased by the presence of additional organic or nutritional material. Simulation analyses based on data from AAPs also indicate that in natural waters the photolysis half-lives for TNT were shorter (3 to 22 hr), while the microbial degradation half-lives were longer (21 to 65 days, including acclimation period). Data generated by Spanggord et al. for 2,4-DNT indicated photolysis half-lives of 1.8 to 6.0 days (Table 8.19) and microbial degradation of 90% by 6 days with a lag period of 2-3 days. The biotransformation rate was much faster for 2,4-DNT than for TNT and indicates little persistence in the aquatic environment.

Confirmation of the low persistence of TNT in the aquatic environment was provided by Jerger et al. (1976). They used sediments from Iowa and Joliet AAPs as microbial sources for degradation studies. In laboratory microcosm studies, Jerger et al. (1976) found that 90% of a 10-ppm TNT solution was transformed following three days of incubation. This is very rapid degradation, and at the 10-100-µg/L concentrations found in receiving streams, it could be even more rapid. Studies by Small (1978), in which he calculated yearly photolytic rate constants for wastewater compounds (Table 8.20), showed that photolysis has a more important effect on some compounds than on others. It is particularly interesting to note the variations in rates between the various dinitrotoluenes.

Persistence of TNT associated munitions in soil and groundwater has been studied less than in aquatic situations, but available data indicate a lengthier persistence. The data are from studies of AAPs, not

TABLE 8.18. SUNLIGHT PHOTOLYSIS OF TNT IN PURE WATER[a]

Latitude (°N)	Summer Half-life (hr)	Winter Half-life (hr)
20	14	22
40	14	45
50	14	84

a. Adapted from Spanggord et al. 1980.

TABLE 8.19. ANNUAL VARIATION OF PHOTOLYSIS HALF-LIFE[a] OF RDX AND 2,4-DNT IN SUNLIGHT DISTILLED WATER[b]

Season	2,4-DNT[c] (N latitude)			RDX[c] (N latitude)		
	20°	40°	50°	20°	40°	50°
Summer	1.8	1.8	1.9	1.1	1.2	1.3
Fall	2.3	3.6	5.4	1.4	2.6	4.4
Winter	2.8	6.0	11.5	1.8	5.0	12.5
Spring	1.9	2.2	2.5	1.2	1.5	2.0

a. Half-lives are in 24-hr days (10 hr sunlight).

b. Adapted from Spanggord et al. 1980.

c. 2,4-DNT = 2,4-dinitrotoluene; RDX = hexahydro-1,3,5-trinitro-1,3,5-triazine.

TABLE 8.20. ENVIRONMENTAL DEGRADATION TESTS OF TNT WASTEWATER COMPOUNDS AND SELECTED RATE CONSTANTS[a]

Compound	Roof-top Test[b]	Photolysis Reactor[c]	Rate Constants, year^{-1}	
			Vaporization	Photolysis
2-Nitrotoluene		X		
4-Nitrotoluene		X		
2,3-Dinitrotoluene	X		30	18
2,4-Dinitrotoluene	X	X	21	28
2,5-Dinitrotoluene	X	X	42	198
2,6-Dinitrotoluene	X	X	49	163
3,4-Dinitrotoluene	X	X	16	4
3,5-Dinitrotoluene	X	X	21	9
1,3-Dinitrobenzene	X	X	16	4
3-Methyl-2-nitrophenol		X		
5-Methyl-2-nitrophenol		X		
2-Amino-4-nitrotoluene		X		
2-Amino-6-nitrotoluene		X		
3-Amino-4-nitrotoluene		X		
3-Amino-2,4-dinitrotoluene	X	X	11	10
3-Amino-2,6-dinitrotoluene	X	X	20	220
4-Amino-3,5-dinitrotoluene	X	X	9	17
4-Amino-2,6-dinitrotoluene	X	X	6	183
5-Amino-2,4-dinitrotoluene	X	X	2	12
1,5-Dimethyl-2,4-dinitrotoluene	X	X	26	64

a. Adapted from Small 1978.

b. Degradation test using beakers exposed to natural sunlight photolysis.

c. Photolysis test using UV light source in laboratory.

laboratory studies. Sanocki et al. (1976) reported the occurrence at Iowa AAP of a former waste lagoon, now filled in with sediments and coal wastes, that contains TNT at levels over 3,000 mg/kg. This site had not been used as a dump for approximately 20 years. The detection of such high levels of TNT after this extended time period suggests that the degradation processes at work in aquatic systems do not function with the same effectiveness in soils. Occurrence of degradation products only at low levels supports the lack of degradation. Keirn et al. (1981) in their review of soil data at Alabama AAP, also found high levels of TNT, dinitrotoluene isomers (by-products from production), and trinitrobenzene more than 35 years after production had ceased. They also commented that the processes that rapidly degrade munitions in aquatic systems do not appear as effective in soil environments.

Only one report (Carpenter et al. 1978) addressed the persistence of TNT and its production wastes in the atmosphere. In general the persistence of the wastes from production processes and incineration is linked to its atmospheric behavior. The particulates, sulfur dioxide (after oxidation and formation to sulfates and sulfuric aerosols), and nitrogen dioxide (after conversion to nitrates and nitric acids) will be deposited on the earth's surface via settling or rain deposition. The carbon monoxide and hydrocarbons will be oxidized to carbon dioxide, water, and aerosols. Ozone will also be formed from the hydrocarbons and nitrogen oxides. The lifetime of a compound in air is dependent on its photodissociation potential and its reactivity with ozone and hydroxyl radicals. The half-lives for TNT associated air pollutants via these processes are given in Table 8.21. The persistence of TNT released into the atmosphere is not addressed directly by Carpenter et al., but it should settle to the ground in the manner of other particulates and could undergo photolysis prior to or after deposition.

8.3.1.2 RDX, HMX, and Associated Wastes

Spanggord et al. (1980) studied the persistence of RDX in aquatic systems in laboratory experiments. They looked at photolytic and microbial degradation under various environmental conditions. Photolysis of RDX does not occur at a fast rate, usually requiring 1.5 to 5 days to degrade 50 percent of a sample. This process varies with latitude and season similar to TNT and 2,4-DNT (Table 8.19). Biodegradation of RDX proceeds even slower. In aerobic conditions, degradation of RDX did not occur after 90 days of incubation with water from the Holston River. Addition of sediment did produce a decrease from 10 ppm to 4 ppm after 36 days (perhaps due to sorption), but two more weeks of incubation produced no further degradation. However, in anaerobic conditions with the presence of extra organic materials, significant degradation occurred, with a 30-ppm solution of RDX reduced to <0.1 ppm after 10 days. This indicates that in most stream systems (aerobic conditions), persistence of RDX would be fairly lengthy. In lake-type aquatic situations (anaerobic conditions), degradation would occur to a greater extent. Simulations with Holston AAP data indicated that photolysis would be the predominant process and half-lives of 1 to 14 days were possible, depending on flow and weather conditions.

TABLE 8.21. HALF-LIVES[a] OF SELECTED AIR CONTAMINANTS ASSOCIATED WITH THE MANUFACTURE OF TNT[b]

Compound (A)	A + hν = $t_{1/2}$[c]	A + O_3 = $t_{1/2}$	A + OH = $t_{1/2}$
Carbon monoxide	-[d]	1 x 10^7 days	4.6-46 days
Methane	-	9.4 x 10^5 days	87-870 days
Toluene	-	330 days	2.1-21 hr
Nitric oxide	-	0.33 min	2.8-28 hr
Nitrogen dioxide	1.1 min	3.0 hr	3.5-35 hr
Nitrous acid	5.8 min	-	7.0-70 hr
Nitric acid	55 hr[e]	-	6.9-69 days
Tetranitromethane	?[f]	?	?
Mononitrotoluenes	?	?	157-1570 days
Sulfur dioxide	102 hr[g]	6 x 10^4 days	26-260 hr
Sulfuric acid	-	-	-

a. The half-life of a reaction is defined by the equation $t_{1/2}$ = 0.693/k sec. The half-lives for each pathway are calculated independently of other processes.

b. Adapted from Carpenter et al. 1978.

c. Photodissociation reaction; hν = light input.

d. A blank space (-) designates that the reaction between a particular pollutant and light, ozone, or hydroxyl radical is unimportant in the atmosphere.

e. Gilbert and Lindsey 1959 as cited in Carpenter et al. 1978.

f. A question mark (?) indicates that the rate constant for the reaction between a pollutant and light, ozone, or hydroxyl radical is unavailable.

g. Badger et al. 1958, as cited in Carpenter et al. 1978.

Information on the persistence of HMX was determined by Spanggord et al. (1982). They found no biodegradation of HMX after 15 weeks in aerobic studies where 4-ppm HMX was added to river water or river water with HMX wasteline sediments. However, HMX was rapidly degraded when extra organic material was added. A 4-ppm concentration was reduced to <0.1 ppm in three days of incubation of yeast-extracted-added water. A similar rate of degradation was observed with water taken below a waste outfall in the Holston River. Under anaerobic conditions, similar results were obtained. No degradation occurred with river water, slow degradation occurred with water and HMX wasteline sediment (reduced from 4 ppm to <0.2 ppm in 91 days), and fast degradation occurred with yeast extract added to water and HMX wasteline sediment (reduced from 4 ppm to <0.1 ppm in 3 days). Studies by Spanggord et al. (1982) on photolysis of HMX indicate that HMX would have a half-life of 4-5 days.

Data for atmospheric persistence of RDX were reported by Carpenter et al. (1978). Basically, the air pollutants resulting from manufacture of RDX would behave like those from TNT manufacture (see Section 8.3.1.1). Again, photodissociation and reaction with ozone and hydroxyl radicals determine the persistence in the atmosphere. Half-lives of air pollutants of RDX manufacture are presented in Table 8.22. The persistence of RDX released into the atmosphere is not addressed directly by Carpenter et al., but RDX should settle to the ground in the manner of other particulates and could undergo photolysis prior to or after deposition.

8.3.2 Bioaccumulation

8.3.2.1 TNT and Associated Wastes

Little information was found on the bioaccumulation of TNT in organisms. Spanggord et al. (1980) tested the biosorption of TNT by bacteria, using both viable and heat-killed cells. They found that most viable cells transformed the TNT rather than accumulated it. The heat-killed cells did demonstrate biosorption but at such a low level as to be insignificant in the environment. A similar study of biosorption of 2,4-DNT by heat-killed cells indicated no significant biosorption. Hartley (1981) studied the bioaccumulation of 2,4-DNT in various organs of bluegills exposed for two weeks. Table 8.23 indicates the rate of uptake, maximum concentration in tissue, bioconcentration factor, and rate of elimination from the studied tissues. He concluded that bioaccumulation was not significant in food chain transfer, since 2,4-DNT was bicconcentrated at low levels and was eliminated within 24 to 72 hours. In a hazard evaluation study of the possible uses for the Alabama AAP, Rosenblatt and Small (1981) calculated some partition coefficients for plant to animal food chain transfers of TNT and several associated compounds. They used a bioconcentration factor (BF) based on the following equation:

$$\log BF = 1.2 - 0.56 \log SS$$

where BF is in milligram substance per kilogram adipose tissue/milligram substance per kilogram food on a dry weight basis, and SS is water solubility in micrograms per liter. The resulting values are given in Table 8.24. The low values for the compounds indicate that bioaccumulation from plants to animals is not a significant problem.

8.3.2.2 RDX and HMX Associated

Spanggord et al. (1980) reported data on biosorption of RDX by viable and heat-killed cells of bacteria. Their findings for RDX were similar to those for TNT and 2,4-DNT: no significant biosorption was noted. In addition, no transformation by the bacteria occurred. The bioaccumulation of RDX in bluegills, channel catfish, and fathead minnows after 28 days of exposure was studied by Bentley et al. (1977a). Bioaccumulation in the visceral and edible tissues of these species occurred and reached a steady state after 14 days of exposure. The uptake and elimination data are presented in Table 8.25 for each species

TABLE 8.22. HALF-LIVES[a] OF SELECTED AIR CONTAMINANTS ASSOCIATED WITH THE MANUFACTURE OF RDX[b]

Compound (A)	A + hν = $t_{1/2}$[c]	A + O_3 = $t_{1/2}$	A + OH = $t_{1/2}$
Methane	-[d]	9.4×10^5 days	87-870 days
Toluene	-	330 days	2.1-21 hr
Carbon monoxide	-	1.0×10^7 days	4.6-46 days
Methanol	-	?[e]	16.5-165 hr
n-Propanol	-	?	4.1-41 hr
n-Butanol	-	6.3×10^3 days	2.3-23 hr
Phenol	?	?	29.5-295 min[f,g]
Formaldehyde	99 min	1.9×10^6 days	1.1-11 hr
Acetaldehyde	12 hr[h]	120 days	46.2-462 min
Acetone	14 hr	?	12-120 hr[g,i]
Methyl ethyl ketone	14 hr	2.2×10^6 days	4.7-47 hr
Cyclohexanone	?	4.2×10^5 days	2.5-25 hr
Formic acid	-	?	2.8-28 days
Acetic acid	-	2.4×10^4 days	20.5-205 days
Methyl formate	-	?	?
Methyl acetate	-	?	?
n-Propyl formate	-	?	?
n-Propyl acetate	-	?	?
Isobutyl acetate	-	?	?
Acetic anhydride[j]	-	?	?
Ammonia	-	?	4.0-40 days
Methylamine	-	?	42.6-426 min
Dimethylamine	-	?	<42-420 min
Nitric oxide	-	0.33 min	2.8-28 hr
Nitrogen dioxide	1.1 min	3.0 hr	3.5-35 hr
Nitrous oxide	-	-	$(1.72\text{-}17.2) \times 10^4$ days
Nitrous acid	5.8 min	-	7.0-70 hr
Nitric acid[k]	55 hr	-	6.9-69 days
Nitromethane	4.3 hr	?	16.5-165 hr
Methyl nitrate	92 hr	-	?
Sulfur dioxide[l]	120 hr	6×10^4 days	26-260 hr

a. The half-life of a reaction is defined by the equation, $t_{1/2} = 0.693/k$ sec. The half-lives for each pathway are calculated independently of other processes.

b. Adapted from Carpenter et al 1978.

c. Photodissociation reaction; hν = light input.

d. A blank space (-) designates that the reaction between a particular pollutant and light, ozone, or hydroxyl radical is unimportant in the atmosphere.

e. A question mark (?) indicates that the rate constant for the reaction between a pollutant and light, ozone, or hydroxyl radical is unavailable.

f. The half-life of the addition reaction between o-cresol and hydroxyl radical.

g. Values are approximations.

h. The rate constant used to determine this half-life is the rate constant for the photolysis of acetaldehyde at a solar zenith angle of 0° (z = 0°).

i. The rate constant between acetone and hydroxyl radicals was estimated; see Carpenter et al. 1978, citing Ambridge et al. 1976.

j. The half-life for the hydrolysis of acetic anhydride is 4.3 min.

k. Carpenter et al. 1978, citing Domerjian et al. 1974.

l. Carpenter et al. 1978, citing Leighton (reference 11).

TABLE 8.23. BIOACCUMULATION DATA OF 2,4-DINITROTOLUENE (2,4-DNT)[a] IN BLUEGILL TISSUE[b]

Tissue	Uptake Rate[c] ± S.D.[e]	Maximum 2,4-DNT Concentration in Tissue[f] ± S.D.	Bioconcentration Factor[g]	Elimination[d] Rate ± S.D.
Whole body	15.36±1.40	72.19±9.57	24.8	24.94±3.78
Brain	43.76±9.52	199.37±12.94	102.8	77.09±5.07
Kidney	23.64±1.93	140.41±10.93	48.25	31.30±4.73
Stomach/Intestine	18.70±4.35	84.98±9.79	29.20	19.96±4.42
Gill	16.40±3.02	68.74±12.41	23.62	19.62±4.71
Liver	14.22±2.11	85.61±8.02	29.41	24.56±1.72
Striated muscle	6.02±0.586	30.82±4.51	10.59	9.53±1.43

a. Exposed to ^{14}C ring labeled 2,4-DNT at 240 nanocuries/liter plus unlabeled carrier at rate of 3.0 mg/L for two weeks.

b. Adapted from Hartley 1981.

c. Rate = micrograms 2,4-DNT uptake per gram of tissue per day.

d. Rate = micrograms 2,4-DNT lost per gram of tissue per day.

e. S.D. = standard deviation.

f. Micrograms 2,4-DNT per gram tissue.

g. Concentration of 2,4-DNT in tissue / concentration 2,4-DNT in the water.

at the two test concentrations used. Elimination of all RDX from the tissues at the low dose exposure occurred after 14 days in the bluegill and catfish but did not occur totally in the minnow. At the higher dose, minnows and catfish eliminated 70 to 84 percent of the accumulated RDX, but no elimination occurred in the bluegill. In conclusion, the authors felt that bioaccumulation was not significant in any of the tested species.

8.3.3 Degradation of Munitions in the Environment

8.3.3.1 Biological Degradation of TNT and Associated Compounds

As previously indicated, TNT and compounds associated with its production can be degraded and transformed by bacterial and fungal species in aquatic and soil environments. The bacterial species that have been identified as effective degraders of TNT are shown in Table 8.26 along with the resulting degradation products. Mechanisms by which the degradation occurs have been proposed by several authors.

It is generally agreed that degradation of TNT occurs fastest in the presence of added nutrients. Glucose added at varying levels resulted in TNT transformation (Osmon and Klausmeier 1972; Won et al. 1974; Weitzel et al. 1975; Jerger et al. 1976; Amerkhanova and Naumova 1978; Hoffsommer et al. 1978). Addition of yeast produced faster transformation of TNT with reported rates of 100 mg/L in 6 days (Osmon and

TABLE 8.24. BIOCONCENTRATION FACTORS[a] FOR SUBSTANCES OF CONCERN AT ALABAMA ARMY MUNITION PLANT[b]

Substance	BF[c]
2,4,6-Trinitrotoluene	2.24×10^{-2} [d]
2,4-Dinitrotoluene	1.43×10^{-2} [d]
Trinitrobenzene	4.76×10^{-2}
1,3-Dinitrobenzene	1.1×10^{-2}
Diphenylamine	4.45×10^{-2} [e]
Aniline	9.4×10^{-4} [e]
N,N-Dimethylaniline	1.46×10^{-3}
Nitrobenzene	3×10^{-2} [f]

a. Bioconcentration factor between plant and animal levels of foodchain.

b. Adapted from Rosenblatt and Small 1981.

c. Bioconcentration factor in $\frac{\text{mg substance/kg adipose}}{\text{mg substance/kg food}}$

d. Based on excretion and metabolism considerations, a plant-to-animal partition coefficient (K_{pa}) as low as 0.1x the tabulated value may be applicable.

e. Based on excretion and metabolism considerations, a K_{pa} as low as 0.5x the tabulated value may be applicable.

f. Value from Geyer et al. 1980, as cited in Rosenblatt and Small 1981.

Klausmeier 1972) and 100 µg/mL in 24 hrs (Won et al. 1974). The transformation rate was also shown to increase with the yeast concentration (Spanggord et al. 1980). Transformation of TNT occurred more slowly when it was the sole source of carbon (Weitzel et al. 1975; Traxler 1975; Spanggord et al. 1980); only by using population densities in excess of 7.5 mg cells/mL (far above normal environmental levels) could rapid transformation rates (90 percent in 24 hours) be achieved (Traxler 1975). The increase in metabolic rate with the addition of nutrients indicates that cometabolism may be occurring in the degradation of TNT.

The extent of reported structural degradation of TNT was found to vary between researchers. Breakdown of the ring structure was not found by some reseachers (Hoffsommer et al. 1978; Isbister et al. 1980; Spanggord et al. 1980), while ring cleavage was indicated by others (Traxler 1975; Hale et al. 1979). Both of these groups used ^{14}C-labels to identify degradation products. Unfortunately, in the studies indicating ring cleavage, the effects of photolysis were unclear. Hale et al. mentioned this uncertainty, but no reference to it was made in the Traxler studies. Utilization of the nitrite groups in TNT by bacteria was more universally accepted (Traxler 1975; Amerkhanova and Naumova 1978; Spanggord et al. 1980).

TABLE 8.25. MEASURED ^{14}C-RESIDUES[a] CALCULATED AS RDX[b] IN THE EDIBLE AND VISCERAL TISSUE OF BLUEGILL (Lepomis macrochirus), CHANNEL CATFISH (Ictalurus punctatus), AND FATHEAD MINNOW (Pimephales promelas)[c]

		Mean Measured ^{14}C-Residues (mg/kg)							
		0.014 mg/L Dose				1.0 mg/L Dose			
Period	Day	Edible Tissue	X[d]	Viscera	X[e]	Edible Tissue	X[d]	Viscera	X[e]
				Bluegill					
Exposure	1	0.029 (0.017)[f]	3.1	0.072 (0.059)	8	2.9 (1.3)	2.9	8.1 (4.1)	8
	3	0.035 (0.011)	3.5	0.095 (0.047)	9.5	4.3 (2.2)	4.3	13 (7)	13
	7	0.038 (0.016)	2.5	0.074 (0.048)	5	4.0 (1.8)	4.0	12 (5)	12
	10	0.048 (0.010)	1.9	0.04 (0.011)	2	4.7 (2.9)	4.7	12 (10)	12
	14	0.14 (0.01)	6.4	0.27 (0.04)	12	2.6 (1.5)	3.8	7.8 (4.1)	11
	21	0.048 (0.007)	4.8	0.13 (0.06)	10	3.0 (0.3)	4.1	7.5 (0.3)	10
	28	0.046 (0.005)	4.7	0.091 (0.014)	9	3.8 (0.1)	3.5	6.8 (1.1)	6
Depuration	1	0.040 (0.003)		0.067 (0.019		3.1 (1.4)		8.4 (4.8)	
	3	0.049 (0.022)		0.083 (0.023)		3.0 (0.8)		3.5 -	
	7	0.033 (0.007)		0.064 (0.002)		1.5 (0.4)		2.7 (0.6)	
	10	0.031 (0.010)		0.034 (0.007)		2.0 (1.5)		4.0 (3.8)	
	14	<0.001		<0.002		3.6 (1.5)		6.4 (3.6)	
				Channel Catfish					
Exposure	1	0.016 (0.002)	1.7	0.027 (0.006)	2.9	1.7 (0.1)	1.7	2.8 (0.3)	2.8
	3	0.019 (0.003)	1.9	0.028 (0.002)	2.8	1.7 (0.3)	1.7	2.9 (0.5)	2.9
	7	0.033 (0.004)	2.0	0.045 (0.002)	3	1.8 (0.1)	1.8	3.2 (0.6)	3.2
	10	0.071 (0.008)	2.8	0.12 (0.02)	4.8	2.0 (0.3)	2.0	3.2 (0.2)	3.2
	14	0.12 (0.01)	5.5	0.12 (0.11)	5.5	0.08 (0.2)	1.2	1.6 (0.4)	2.3
	21	0.032 (0.004)	3.2	0.060 (0.005)	6	2.5 (0.3)	3.4	4.1 (1.1)	5.5
	28	0.039 (0.003)	4.0	0.049 (0.008)	5	3.2 (0.1)	2.9	3.6 (0.2)	3.3
Depuration	1	0.020 (0.002)		0.030 (0.006)		0.96 (0.19)		1.8 (0.3)	
		0.017 (0.001)		0.024 (0.006)		0.78 (0.15)		1.0 (0.4)	
	7	0.017 (0.004)		0.026 (0.006)		0.65 (0.16)		0.78 (0.15)	
	10	0.013 (0.002)		0.015 (0.002)		0.66 (0.06)		0.74 (0.08)	
	14	<0.002		<0.002		0.56 (0.12)		0.47 (0.27)	
				Fathead Minnow					
Exposure	1	0.013 (0.003)	1.4	0.023 (0.002)	2.5	1.4 (0.2)	1.4	2.2 (0.2)	2.2
	3	0.018 (0.004)	1.8	0.033 (0.003)	3.3	2.0 (0.1)	2.0	3.8 (0.5)	3.8
	7	0.031 (0.008)	2.1	0.047 (0.005)	3.2	1.8 (0.2)	1.8	3.4 (0.1)	3.4
	10	0.074 (0.004)	3.0	0.15 (0.01)	6	2.1 (0.3)	2.1	4.1 (0.6)	4.1
	14	0.13 (0.01)	5.9	0.26 (0.03)	12	1.4 (0.5)	3.8	3.2 (0.8)	4.6
	21	0.047 (0.007)	4.7	0.047 (0.011)	10	3.2 (0.7)	4.2	7.7 (0.3)	10
	28	0.058 (0.012)	5.9	0.11 (0.02)	11	4.4 (0.4)	4.0	9.6 (0.7)	8.8
Depuration	1	0.037 (0.011)		0.070 (0.006)		2.2 (1.2)		4.5 (0.1)	
	3	0.023 (0.004)		0.071 (0.043)		2.1 (0.4)		3.8 (0.4)	
	7	0.028 (0.012)		0.050 (0.005)		1.8 (0.5)		3.4 (0.6)	
	10	0.029 (0.008)		0.038 (0.005)		2.2 (1.0)		3.2 (0.3)	
	14	0.029 (0.007)		0.032 (0.006)		1.3 (0.9)		1.6 (0.5)	

a. Residues resulted from 28 days of continuous exposure to ^{14}C-RDX concentrations of 0.014 and 1.0 mg/L and from a 14-day period of depuration.

b. RDX = hexahydro-1,3,5-trinitro-1,3,5-triazine.

c. Adapted from Bentley et al. 1977a.

d. Bioconcentration factor in edible tissue based on the mean concentration of ^{14}C-RDX in water of the previous day and that day.

e. Bioconcentration factor in nonedible tissue based on the mean concentration of ^{14}C-RDX in water of the previous day and that day.

f. Mean and standard deviation.

TABLE 8.26. BACTERIAL SPECIES AND METABOLITES IN BIODEGRADATION STUDIES OF TNT AND ASSOCIATED COMPOUNDS

Source Munition[a]	Species Name	Metabolites Identified[a]	Reference
Synthetic[b]	Achromobacter agilis	NG	Bringmann and Kuehn 1971
TNT	Pseudomonas aeruginosa	NG	Osmon and Klausmeier 1972
TNT	Pseudomonas spp.	4-AZ, 6-AZ, 2-AM, 4-OH, DiAm[c]	Won et al. 1974
TNT	Pseudomonas sp. I	NG	Amerkhanova and Naumova 1975
TNT	Pseudomonas striata	NG	Amerkhanova and Naumova 1975
TNT	Escherichia coli	NG	Amerkhanova and Naumova 1975
TNT	Sediment Population[c,d]	OH-amino-DNT	Weitzel et al. 1975
TNT	Sediment Population[c,e]	OH-amino-DNT	Jerger et al. 1976
TNT	Escherichia coli	4,4'-AZ[f], 2,4-AM[g]	McCormick et al. 1976
TNT	Veillonella alkalescens	2,4-AM	McCormick et al. 1976
TNT	Clostridium plasterianum	2-OH	McCormick et al. 1976
TNT	Pseudomonas FR2	4,4'-AZ, 4A, 2,4-AM	McCormick et al. 1976
TNT	Bacillus subtilis	hydroxylaminotoluene, aminotoluene	Klausmeier et al. 1976
TNT	Pseudonomas aeruginosa	hydroxylaminotoluene, aminotoluene	Klausmeier et al. 1976
TNT	Pseudonomas denitrificans	NG	Amerkhanova and Naumova 1978
TNT	Escherichia coli	NG	Amerkhanova and Naumova 1978
TNT	Bacillus sp.	NG	Yang et al. 1979
TNT	Citrobacter sp.	NG	Yang et al. 1979
TNT	Enterobacter sp.	NG	Yang et al. 1979
TNT	Klebsiella sp.	NG	Yang et al. 1979
TNT	Escherichia sp.	NG	Yang et al. 1979
TNT	Soil populations	4-amino-DNT	Osmon and Andrews 1978
TNT	Escherichia coli	4A, 2-AM	Hudock 1972[h]
TNT	Pseudonomas putida	4A, 2-AM	Hudock 1972[h]
TNT	Pseudonomas fluorescens	4A, 2-AM	Hudock 1972[h]
TNT	Pseudonomas denitrificans	4A, 2-AM	Hudock 1972[h]
TNT	Sludge population[i]	4A, 2-AM, 2,4-AM, 2,6-AM	Hoffsommer et al. 1978
TNT	Sediment population[k]	2-AM, 4A, 2,4-AM, 2,6-AM, 4,4'-AZ, 2,2'-AZ	Spanggord et al. 1980.
2,4-DNT	Mucrosporium sp.	2A4NT, 4A2NT, 2,2-DNT, 4,4-DNT, 4-ACE	McCormick et al. 1978[j]
2,4-DNT	Sediment population[k]	CO^2, 4A2NT[c]	Spanggord et al. 1980.
1,3-DNB	Sediment population[k]	CO^2	Mitchell and Dennis 1982

a. TNT = 2,4,6-trinitrotoluene; NG = not given; 4-AZ = 2,2',4,4'-tetranitro-4-azoxytoluene; 6-AZ = 2,2',4,4'-tetranitro-6-azoxytoluene; 2-AM = 4,6-dinitro-2-aminotoluene; 2-OH = 2,6-dinitro-4-hydroxylaminotoluene; DiAM = nitrodiaminotoluene; 2,4-AM = 2,4-diamino-6-nitrotoluene; 4,4'-AZ = 2,2',6,6'-tetranitro-4,4'-azoxytoluene; 4A = 4-amino-2,6-dinitrotoluene; 2,6-AM = 2,6-diamino-4-nitrotoluene; 2A4NT = 2-amino-4-nitrotoluene; 4A2NT = 4-amino-2-nitrotoluene; 2,2-DNT = 2,2'-dinitro-4'-azoxytoluene; 4,4-DNT = 4,4'-dinitro-2,2'-azoxytoluene; 4-ACE = 4-acetamido-2-nitrotoluene; 2,2'-AZ = 4,4',6,6'-tetranitro-2,2'-azoxytoluene; 2,4-DNT = 2,4-dinitrotoluene; 1,3-DNB = 1,3-dinitrobenzene.

b. Synthetic wastewater contained 2,4,6-trinitrotoluene, 2,4-dinitrotoluene, 2,6-dinitrotoluene, 2-nitrotoluene, 4-nitrotoluene, 1,3,5-trinitrobenzene, m-dinitrobenzene, and nitrobenzene.

c. Intermediate metabolites.

d. Aerobic bacterial population from sediments at Iowa AAP.

e. Aerobic bacterial population from sediments at Iowa and Joliet AAPs.

f. Aerobic and anaerobic conditions.

g. Aerobic conditions only.

h. As cited in Hoffsommer et al. 1978.

i. Bacterial population from an aerobic activated sludge source.

j. As cited in Isbister et al. 1980.

k. Aerobic sediment population from Waconda Bay downstream from Volunteer AAP.

The pathways for degradation of TNT have been discussed by several authors. Won et al. (1974) found that TNT is reduced within 24 hours to form 2,2',6,6'-tetranitro-4-azoxytoluene, 2,2'4,4'-tetranitro-6-azoxytoluene, 2,6-dinitro-4-hydroxylaminotoluene and its isomer 2,4-dinitro-6-hydroxylaminotoluene. After depletion of TNT, the azoxy compounds degrade. The other two compounds are then reduced to the respective 4-amino and 2-amino compounds. These are further reduced to nitrodiaminotoluene. McCormick et al. (1976) elaborated these pathways, suggesting (1) the reduction of a nitro group to an amino group followed by oxidative deamination to a phenol, releasing ammonia, and (2) release of a nitro group as nitrite with the formation of a phenol. Figure 8.2 illustrates these pathways. Hoffsommer et al. (1978) reported the metabolic reduction possibilities of TNT in an open treatment ditch. They found evidence for the stepwise nitro reduction process outlined by McCormick et al., including the formation of the tetranitroazoxy compounds. Hoffsommer et al. also found formation of insoluble ^{14}C-labeled products that indicate the presence of 2,4,6-triaminotoluene. Spanggord et al. (1980) supported these proposed degradation pathways and indicated that there is no reduction preference between either the 2- or the 4- position, so there should be equal amounts of the aminodinitrotoluene isomers and the nitroazxoytoluenes.

Microbial degradation of compounds associated with TNT manufacture was also reported. Spanggord et al. (1980) found that 2,4-DNT was rapidly degraded to CO_2 with or without additional nutrient sources. This included ring cleavage. The degradation of 2,6-DNT was not supported in the literature; Spanggord et al. indicated that it was not degraded and accumulated in the test flasks used in the 2,4-DNT studies. Mitchell and Dennis (1982) found that 1,3-dinitrobenzene was capable of being degraded as a sole source of carbon if microbes were used that had been taken from sites associated with TNT production.

Biological reduction of TNT and 2,4-DNT was reported by Parrish (1977) for 190 species of fungi. He found that with glucose added, 183 species could reduce TNT but only five could reduce 2,4-DNT. The transformation products of TNT that were identified included 4-amino-2,6-dinitrotoluene, 4-hydroxylamino-2,6-dinitrotoluene, and 2,2',6,6'-tetranitro-4,4'-azoxytoluene. Using labeled TNT he could not find evidence of ring cleavage.

8.3.3.2 Biodegradation of RDX and HMX

The majority of available data indicate that RDX and HMX cannot be degraded when they are the sole source of carbon or, for RDX, if aerobic conditions are present (Osmon and Klausmeier 1972; Hoffsommer et al. 1978; Spanggord et al. 1980; McCormick et al. 1981). However, when exposed to anaerobic sediment-populations of bacteria and extra nutrients, RDX and HMX were reduced or transformed within 4 to 38 days (Isbister et al. 1980, citing data of Natick 1980 and of Sikka et al. 1980; Spanggord et al. 1980; McCormick et al. 1981). Bacteria that degrade RDX were identified as Pseudomonas spp., Alcaligenes spp., Thiorhodaceae (photosynthetic), and Athiorhodaceae (photosynthetic)

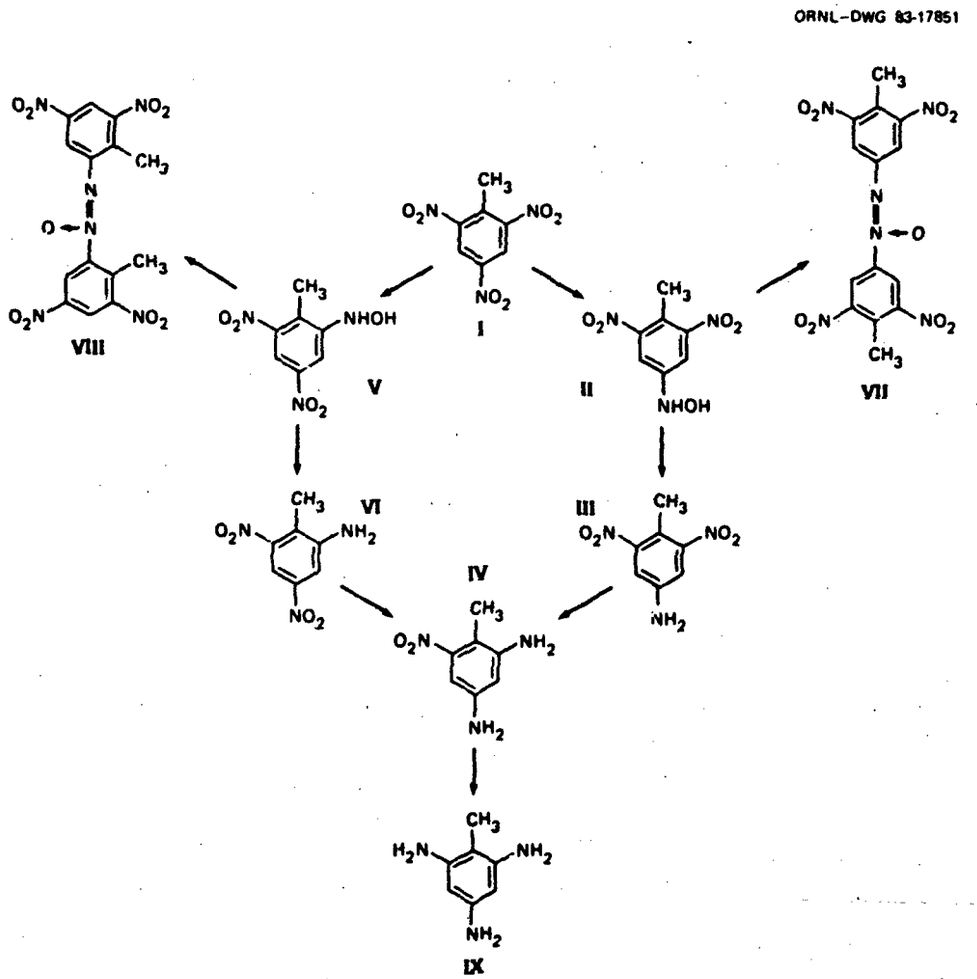

Figure 8.2. Proposed pathways for transformation of TNT by reduction of the nitro groups. Compounds illustrated are: I = 2,4,6-trinitrotoluene; II = 4-hydroxylamino-2,6-dinitrotoluene; III = 4-amino-2,6-dinitrotoluene; IV = 2,4-diamino-6-nitrotoluene; V = 2-hydroxylamino-4,6-dinitrotoluene; VI = 2-amino-4,6-dinitrotoluene; VII = 2,2,6,6'-tetranitro-4,4'-azoxytoluene; VIII = 4,4',6,6'-tetranitro-2,2'-azoxytoluene; and IX = 2,4,6-triaminotoluene. From McCormick et al. 1976.

(Soli 1973; Sullivan et al. 1979, citing data of Green 1972). Transformation products were identified including formaldehyde, methanol, hydrazine, trinitrosotriazine, and a series of RDX nitroso reduction products (Isbister et al. 1980, citing data of Natick 1980; McCormick et al. 1981). Cleavage of the benzene ring in ^{14}C RDX was indicated in soil composting studies (Hale et al. 1979) and in anaerobic sediments mixed with water (Isbister et al. 1980, citing data of Sikka et al. 1980; McCormick et al. 1981). The study by McCormick et al. also identified potential pathways for the anaerobic degradation of RDX (Figure 8.3) with three pathways for cleavage of ring compounds. These compounds are further reduced to give the eventual product of methanol (Figure 8.4). However, in some studies the transformation products did not include ^{14}C labels, suggesting that the RDX does not always degrade totally (Isbister et al. 1980, citing data of Sikka et al. 1980).

Recent information on the microbial degradation of HMX was determined by Spanggord et al. (1982). They found degradation of HMX in aerobic studies where 4 ppm HMX and yeast extract was added to river water or river water with HMX wasteline sediments. The 4-ppm concentration was reduced to <0.1 ppm in three days of incubation of yeast-extracted-added water. A similar rate of degradation was observed with water taken below a waste outfall in the Holston River. Apparently, microorganisms are present in these materials that can aerobically degrade HMX. Under anaerobic conditions, similar results were obtained. No degradation occurred with river water, slow degradation occurred with water and HMX wasteline sediment (reduced from 4 ppm to <0.2 ppm in 91 days), and fast degradation occurred with yeast extract added to water and HMX wasteline sediment (reduced from 4 ppm to <0.1 ppm in 3 days).

8.3.3.3 Physical Degradation of TNT and Associated Compounds

The primary physical mechanism that degrades TNT munitions is photolysis. Exposure to sunlight or any source of ultraviolet (uv) light produces fairly rapid breakdown of the TNT and its associated compounds, including impurities and resulting degradation products. Generally, UV light with a wavelength below 420 nm will degrade TNT directly. Wavelengths between 400 and 500 nm will degrade TNT indirectly through solar-absorption in natural products and the resulting photoreactions (Spanggord et al. 1980).

Andrews and Osmon (1976) found that 1,000 ppm of 1,3,5-TNT in aqueous solution was reduced to 170 ppb after 24 hours exposure to 240-260 nm UV light. This produced a light pink water residual, but ring cleavage to CO_2 and volatile organics was shown. They also found that 2,4- and 2,6-DNT were totally degraded. A pink water solution containing 221 ppm of TNT and 148 ppm of RDX was also degraded after 49 hours under optimum UV-exposure conditions. Spanggord et al. (1980) found that the half-lives of TNT exposed to sunlight in natural water ranged from 3 to 22 hours, and photolysis rate constants ranged from 28 to 40 $days^{-1}$. Photolysis rate constants for other TNT associated compounds were reported by Small (1978) and are shown in Table 8.27.

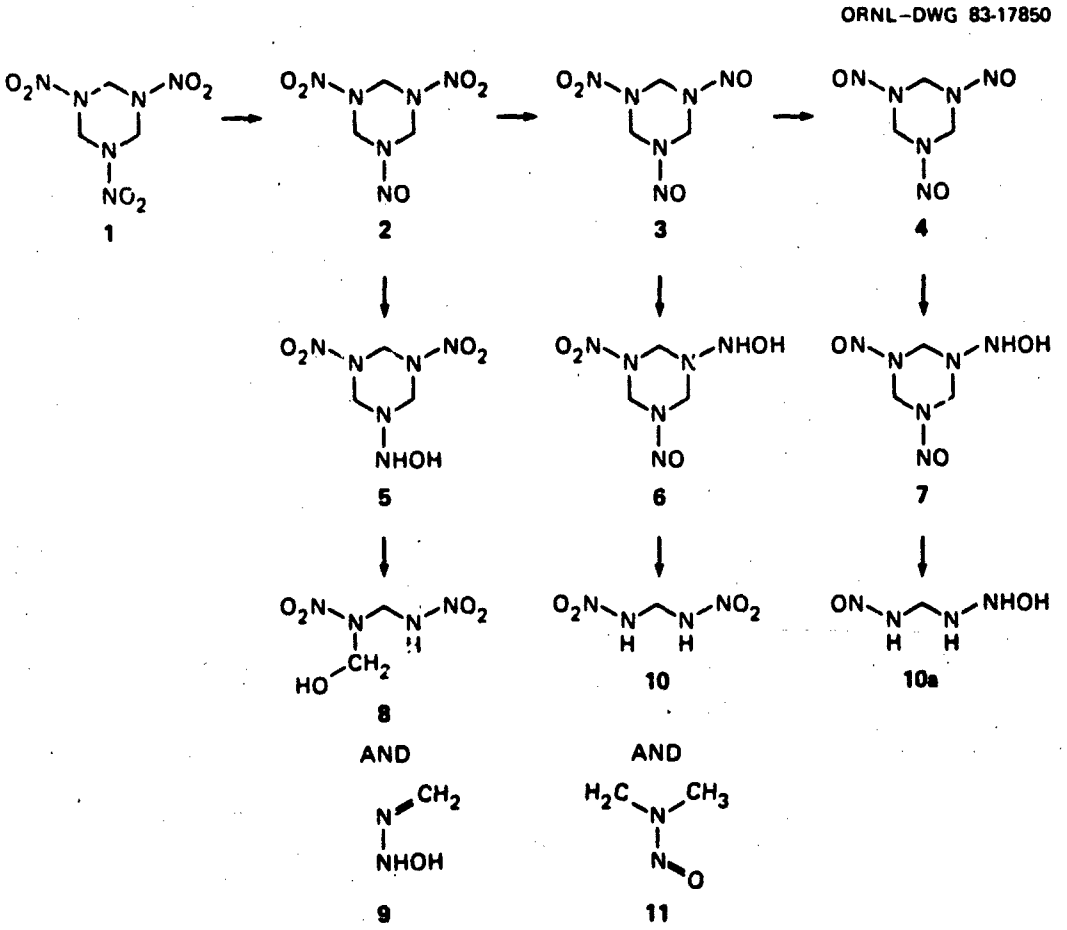

Figure 8.3. Proposed anaerobic degradation pathway of RDX. Compounds are: 1 = RDX (hexahydro-1,3,5-trinitro-1,3,5-triazine); 2 = MNX (hexahydro-1-nitroso-3,5-dinitro-1,3,5-triazine); 3 = DNX (hexahydro-1,3-dinitroso-5-nitro-1,3,5-triazine); 2,4 = TNX (hexahydro-1,3,5-trinitroso-1,3,5-triazine); 5 = 1-hydroxylamino-3,5-dinitro-1,3,5-triazine; 6 = 1-hydroxylamino-3-nitroso-5-nitro-1,3,5-triazine; 7 = hydroxylamino-3,5-dinitroso-1,3,5-triazine; 8 = N-hydroxymethylmethylene-dinitramine; 9 = N-hydroxymethylhydra-zine; 10 = N-hydroxyl-amino-N'-nitromethylenediamine; 10a = N-hydroxyl-amino-N'-nitroso-methylenediamine; and 11 = dimethylnitrosamine radical. From McCormick et al. 1981.

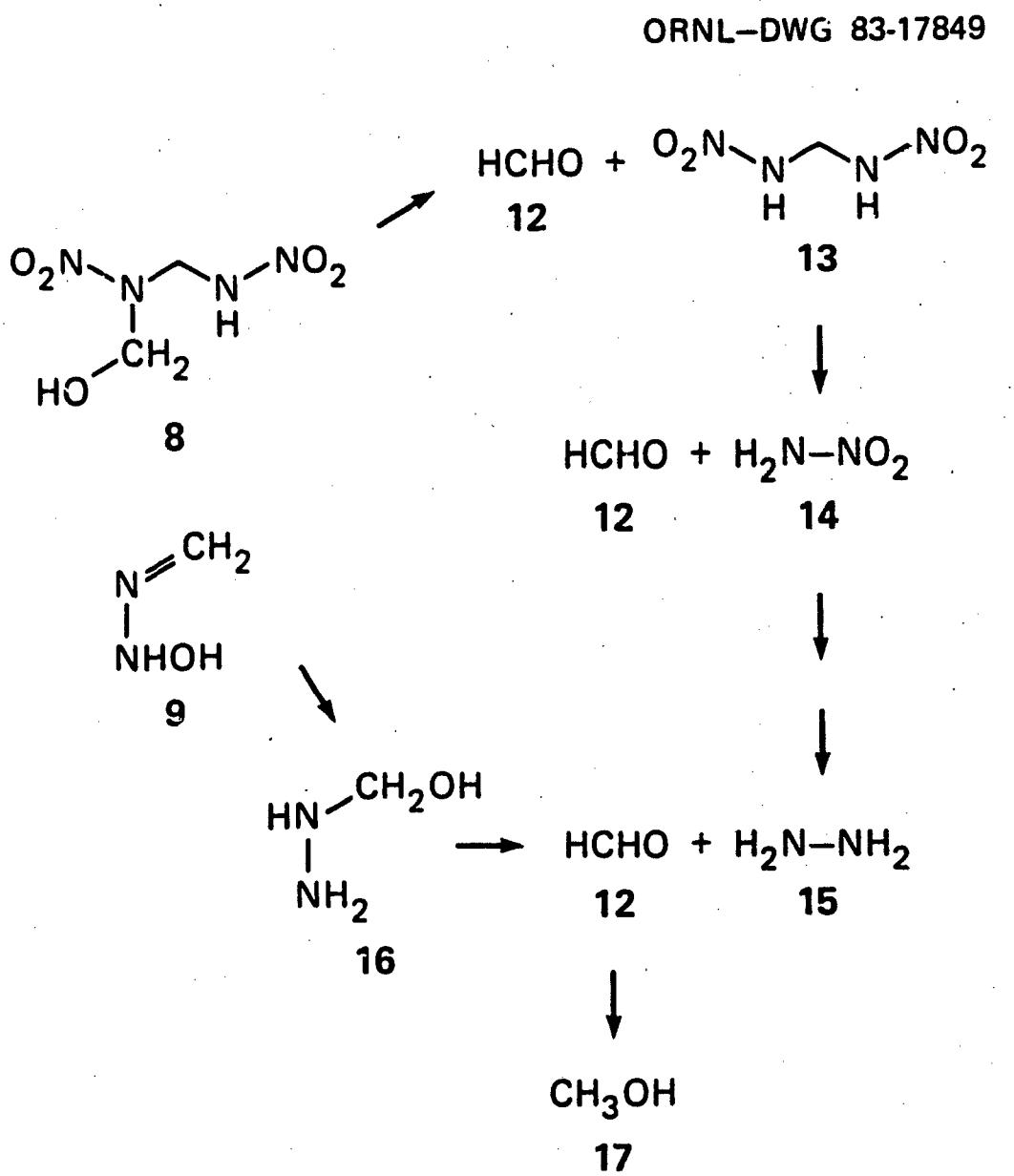

Figure 8.4. Final stages of proposed anaerobic degradation pathway of RDX-derived compounds. Compounds are: 8 = N-hydroxymethyl-methylenedinitramine; 9 = N-hydroxymethylene-hydrazone; 12 = HCHO; 13 = methylenedinitramine; 14 = nitramide; 15 = hydrazine; 16 = hydroxymethylhydrazine; and 17 = methanol. From McCormick et al. 1981.

TABLE 8.27. PHOTOLYSIS RATE CONSTANTS FOR TNT[a] WASTEWATER COMPOUNDS[b]

Compound	Rate constants ($year^{-1}$)
2,3-Dinitrotoluene	18
2,4-Dinitrotoluene	28
2,5-Dinitrotoluene	198
2,6-Dinitrotoluene	163
3,4-Dinitrotoluene	4
3,5-Dinitrotoluene	9
1,3-Dinitrobenzene	4
3-Amino-2,4-dinitrotoluene	10
3-Amino-2,6-dinitrotoluene	220
4-Amino-3,5-dinitrotoluene	17
4-Amino-2,6-dinitrotoluene	183
5-Amino-2,4-dinitrotoluene	12
1,5-Dimethyl-2,4-dinitrotoluene	64

a. TNT = 2,4,6-trinitrotoluene.

b. Adapted from Small 1978.

The conditions in which aqueous solutions of TNT are exposed to sunlight do effect the photolysis process. Solutions that contain organic matter result in faster photolysis rates than solutions in distilled water (Spanggord et al. 1980). The pH of the solution can be a factor in the rate of photolysis if the water contains little organic matter (Isbister et al. 1980, citing data of Burlinson et al. 1973; Spanggord et al. 1980) and, generally, lower rates are produced at lower pHs. The rate also increases with time, due to the increase in photolysis products in solution (Spanggord et al. 1980).

The pathway by which TNT is reduced has not been fully elucidated (Mabey et al. 1983; Spanggord et al. 1980). Intermediate compounds have been identified and attributed to photolysis (Table 8.28), but the details of their formation are only generally known. Several authors have suggested that the principal information supports a degradation process via a triplet-sensitized mechanism (Rosenblatt et al. 1971; Spanggord et al. 1980; Mabey et al. 1983). This mechanism postulates formation of a triplet exited state of the TNT compound similar to the one in Figure 8.5, which permits a weak nucleophile to attack and remove a NO_2 or X^- group (Rosenblatt et al. 1971). Another proposed mechanism is based on the formation of a complex between humic substances in water

TABLE 8.28. KNOWN PHOTOLYSIS PRODUCTS OF 2,4,6-TNT[a]

1,3,5-Trinitrobenene
1,3-Dinitrobenzene
2,4-Dinitrotoluene
2,4,6-Trinitrobenzaldehyde
2,4,6-Trinitrobenzyl alcohol
2,4,6-Trinitrobenzonitrile
2,4,6-Trinitrobenzaldoxime
4,6-Dinitro (1,2) benzisoxazole
4,6-Dinitrophenol
2,4,6-Trinitrobenzoic acid
2-Amino-4,6-dinirobenzoic acid
2,2'-Dicarboxy-3,3',5,5'-tetranitro-azoxybenzene
2,2'-Dicarboxy-3,3',5,5'-tetranitro-azobenzene
2-Carboxy-3,3',5,5'-tetranitro-azoxybenzene
N-(2-Carboxy-3,5-dinitrophenyl)-2,4,6-trinitrobenzamide

a. Adapted from Jerger et al. 1976.

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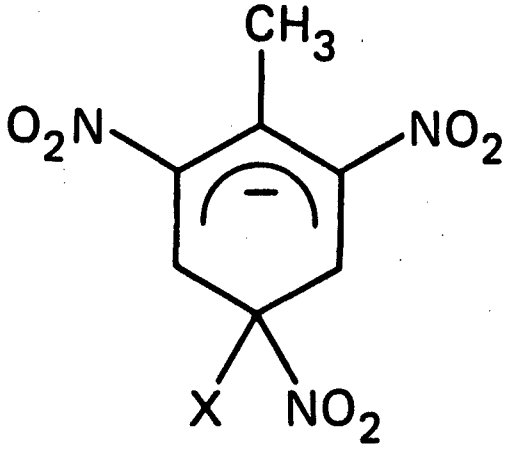

Figure 8.5. Proposed excited state intermediate of TNT found in photolytic reactions. From Rosenblatt et al. 1971.

and the TNT compound (Spanggord et al. 1981). The presence of such a complex would alter the UV absorbances responsible for photodegradation of TNT. Spanggord et al. (1981) tested the possibilty of a TNT-humic acid complex by using a polargraphic technique to determine if the "free" TNT in pure water had a different reduction potential than TNT in water from Searsville pond (high in organic substances). They did find differences in the reduction polargrams between the two TNT/water combinations, indicating the possible formation of a TNT-humic acid complex. Theoretically, such a complex might absorb sunlight more readily than TNT alone, leading to faster photodegradation.

Information on photolytic processes occurring in soils was reported by Keirn et al. (1981). In a review of Alabama AAP, they noted that some photolysis of TNT in soil must occur due to high levels of trinitrobenzene found in surface layers. They also proposed several photolytic pathways for conversion of TNT and DNT compounds in soils to tri- or dinitrobenzaldehyde compounds, respectively.

The effects of photolysis on airborne by-products of TNT manufacture were discussed by Carpenter et al. (1978). Rapid photodissociation (within minutes to several hours) was found for the by-products nitrous acid, nitromethane, formaldehyde, acetone, and nitrogen dioxide. Photodissociation was much slower (several days) for nitric acid and methyl nitrate. However, Carpenter et al. (1978) concluded that for the 2,4-di-, 2,6-di-, and 2,4,6-tri-nitrotoluenes, photolytic reactions played a minor part in the atmospheric chemistry associated with TNT production. Reactions of a nonphotolytic nature also play a large role in degrading TNT by-products. Reactions with hydroxyl radicals and ozone can act as swiftly as photolysis. In general the atmospheric reactions interact in very complex behaviors depending on compound concentrations, time of day, and meteorological conditions. A detailed description of the various processes is given in Carpenter et al. 1978, Volume 3.

8.3.3.4 Physical Degradation of RDX and HMX

As with TNT, the primary physical mechanism that degrades RDX and HMX in aqueous solutions is photolysis. The range of UV wavelengths that causes photolytic reactions with these munitions is similar to that of TNT, generally between 240 and 350 nm. Andrews and Osmon (1976) found that at 240-260 nm, RDX and HMX were totally degraded after 312 hours. In a maximum exposure situation, 45 ppm of RDX and 5 ppm of HMX were reduced without any TLC-detectable products after two hours. Jain (1976) reported that a 4.5 mg/L solution of HMX was degraded after 30 minutes exposure to UV light and ozone. Spanggord et al. (1982) found that 0.5 ppm of HMX had a half-life of 4-5 days when exposed to natural sunlight. Rapid rates of disappearance (50 mg/L in 10 minutes) were found by Kubose and Hoffsommer (1977) and by Smetana and Bulusu (1977) for a 254-nm wavelength. However, at 350 nm the same degree of degradation took three times as long (Smetana and Bulusu 1977). Exposure of solid RDX to 366 nm failed to cause any degradation, and no gas evolved. Spanggord et al. (1980) found half-lives of 1 to 14 days for RDX exposed

to 313 nm. This rate was the same in either distilled water or natural water, indicating that no indirect photolytic effect occurs. It was also shown that the pH of the solution did not affect the rate of photolysis, but the solution did drop in pH to 3-4 after photolysis (Kubose and Hoffsommer 1977).

The degradation pathway for RDX when exposed to UV light was also discussed by Kubose and Hoffsommer (1977). They found two possible routes of decomposition depending on the UV wavelengths. At levels greater than 280 nm, the principal step is the homolytic cleavage of the nitramine bond to give an azayl radical and NO_2. When this occurs in acidic conditions, NO is formed and reacts with azayl radical to form a mono-nitroso analog of RDX (they proposed 1-nitroso-3,5-dinitro-1,3,5-triazacyclohexane). When the wavelengths are near 220 nm, the primary step is cleavage of N-O bond of nitro group to form the nitroso analog directly. This reaction is independent of pH. Other metabolites formed, and their mole ratios to RDX, are given in Table 8.29. The presence of formaldehyde, ammonia, and the other gases indicates that the ring is broken in the final degradation steps. Smetana and Bulusu (1977) also found these final metabolite products. Spanggord et al. (1980) identified many of these products including formaldehyde and other gases. However, they were unable to confirm the presence of the nitroso analog found by Kubose and Hoffsommer.

TABLE 8.29. MOLE RATIO OF PRODUCTS TO PHOTOLYZED RDX[a,b]

Wavelength (λ)	$\frac{NO_3^-}{RDX}$	$\frac{NO_2^-}{RDX}$	$\frac{CH_2}{RDX}$	$\frac{NH_3}{RDX}$	$\frac{N_2O}{RDX}$	$\frac{N_2}{RDX}$
>220 nm	trace	2.4	0.8	0.6	0.05-0.1	0.1-0.2
>280 nm	0.7	2.0	0.6	0.7	—	—

a. RDX = hexahydro-1,3,5-trinitro-1,3,5-triazine.

b. Adapted from Kubose and Hoffsommer 1977.

Other physical methods of degradation were mentioned including hydrolysis, thermal decomposition, and reactions with ozone. Hydrolysis and ozone reactions were much slower than photolysis and only reduced 50 percent of the RDX at best (Smetana and Bulusu 1977; Sullivan et al. 1979, citing data of Hoffsommer and Rosen 1973). Thermal decomposition of RDX was quite similar to photolysis, reducing 97 percent of an RDX solution. Also the decompostion products were similar, with CO_2, N_2O, N_2, NO, CO, HCN, HC_2O, and H_2O found (Smetana and Bulusu 1977).

Data on the physical degradation of atmospheric released by-products and munition compounds associated with the production of RDX and HMX are treated by Carpenter et al. (1978). They found essentially the same reactions and behaviors for these compounds as for TNT-related releases (see Section 8.3.3.3).

8.4 EFFECTS ON HABITATS

8.4.1 Water Quality

8.4.1.1 TNT and Associated Wastes

Information on the effects of TNT on water quality consists primarily of data generated from various AAP sites. Most of these studies measured various physical and chemical parameters of the affected aquatic systems and related the changes to distance from the AAP or to actual levels of TNT in the water or sediments. These data are quite variable because many factors affect the water quality, including the munition production levels and conditions, weather conditions, and time of year.

A study of the water quality at Radford AAP indicated that some parameters were elevated in the river below the waste discharge point (Huff et al. 1975a). The affected parameters included total solids, chemical oxygen demand (COD), alkalinity, color, total dissolved solids (TDS), orthophosphates, total organic carbon (TOC), sulfates, nitrates, nitrites, total volatile solids, and total Kjedahl nitrogen (TKN). The changes in parameter values are shown in Table 8.30 and occur at or below the discharge points (stations E and F in the table). However, the authors cautioned that the data were merely indicative and not conclusive of an association between TNT and a decrease in water quality.

A more conclusive link between changes in water quality and TNT levels in aquatic systems was found in several studies of the Volunteer AAP (Huff et al. 1975b; Sullivan et al. 1977). In 1974, Huff et al. (1975b) found several parameters in water column analyses that demonstrated higher values near the waste discharge point of VAAP in Waconda Bay (Table 8.31). In comparison with established standards, only the elevated nitrate/nitrite levels were considered by the authors to pose a problem. The measured concentrations approach the LC_{50} values for juvenile rainbow trout, and the high levels would also result in a stimulation of aquatic growth due to nitrogen's nutrient value. Analyses of sediments for affected parameters were not conclusive. A more thorough study in 1975 also indicated an association between high values and proximity to the VAAP (Sullivan et al. 1977). They found that the parameters of specific conductance, total hardness, TDS, and concentrations of sulfate, chloride, ammonia, nitrate, nitrite, and TKN were related to distance from the VAAP discharge (Table 8.32). They also found that levels of TNT and its by-products generally were higher in stations near the VAAP, although no consistent gradient was observed. Analyses of sediments in Waconda Bay further supported this association.

TABLE 8.30. WATER ANALYSES OF RADFORD ARMY AMMUNITION PLANT, NEW RIVER[a]

Parameters	Stations[b]				
	A	E	F	J	K
Alkalinity (ppm $CaCO_3$)	35	45	100	42	42
Color (chloroplatinate units)	8	8	65	13	18
Sulfates (ppm SO_4)	5	145	63	10	17
Total solids (ppm)	47	528	351	71	68
Total dissolved solids (ppm)	47	528	350	71	68
Total suspended solids (ppm)	<1	<1	1	<1	<1
Total volatile solids (ppm)	23	197	84	28	19
Nitrates (ppm N)	0.42	2.08	3.52	0.96	1.74
Nitrites (ppm N)	<0.05	0.66	0.53	0.06	0.09
Total Kjeldahl nitrogen (ppm N)	1.44	1.79	2.39	1.20	1.31
Ammonia (ppm N)	<0.25	<0.25	<0.25	<0.25	<0.25
Orthophosphates (ppm P)	<0.01	0.15	1.12	<0.01	<0.01
Chemical oxygen demand (ppm)	52.8	48.4	132.0	48.4	47.1
Total organic carbon (ppm)	7	-	33	14	25
2,4,6-Trinitrotoluene (ppm)	<0.1	<0.1	<0.1	<0.1	<0.1
Trinitrobenzene (ppm)	<0.2	<0.2	<0.2	<0.2	<0.1
2,4-Dinitrotoluene (ppm)	<0.1	(0.10)	(0.55)	<0.1	<0.1
2,6-Dinitrotoluene (ppm)	<0.1	<0.1	<0.1	<0.1	<0.1

a. Adapted from Huff et al. 1975a.

b. Station A, E, and F were sampled on September 18, 1974; stations J and K were sampled on September 19, 1974; Station A is upstream control; Station E is below neutralized acid effluent input; Station F is below Stroubles Creek entrance; Station J is below loading and packing plant; Station K is at downstream plant boundary.

Table 8.33 shows that the upper end of Waconda Bay has higher values of volatile solids, COD, nitrate-nitrogen, and total phosphorus, as well as higher contents of TNT.

Holston AAP was examined for impacts on water quality in a study by Huff et al. (1975c). Huff and his coworkers found only a few parameters that were affected by discharges from Holston AAP (Table 8.34). The parameters of sulfates, total solids, TDS, total suspended solids, COD, and TOC showed increases related to the proximity to Holston AAP. The impact of the AAP on water quality was, however, masked by the effects of other industrial discharges located upstream.

The effect of TNT waste discharges from Joliet AAP on water quality was reported in three studies (Ghassemi et al. 1976; Stilwell et al. 1976; Jerger et al. 1976). The report by Ghassemi et al. (1976) discussed data generated by earlier researchers in 1967. Table 8.35 contains the parameters showing the most significant changes. The sites

TABLE 8.31. WATER QUALITY DATA FOR THE VOLUNTEER ARMY AMMUNITION PLANT-ASSOCIATED WACONDA AND CONTROL BAYS, CHICKAMAUGA LAKE[a]

Parameters (units)	Stations[b]			
	A2	B2	E2	X2
Color (CP units)	27	23	7	6
Total sulfates (ppm SO_4)	72	37	17	15
Total solids (ppm)	335	179	93	129
Total dissolved solids (ppm)	334	179	93	129
Total volatile solids (ppm)	169	61	64	76
Nitrates (ppm N)	2.32	3.49	1.48	0.42
Nitrites (ppm N)	0.73	0.39	0.07	<0.05
Total Kjeldahl nitrogen (ppm N)	0.27	1.00	0.66	0.58
Chemical oxygen demand (ppm)	23.8	26.4	72.6	19.8
Total organic carbon (ppm)	62	7	11	2
2,4,6-Trinitrotoluene (ppm)	<0.1	<0.1	<0.1	<0.1
Trinitrobenzene (ppm)	<0.2	<0.2	<0.2	<0.2
2,4-Dinitrotoluene (ppm)	<0.1	<0.1	<0.1	<0.1
2,6-Dinitrotoluene (ppm)	<0.1	<0.1	<0.1	<0.1

a. Adapted from Huff et al. 1975b.

b. Station A2 was at VAAP waste discharge point, B2 was 500 m from discharge point, E2 was 3000 m from discharge point, and X2 was in a reference bay not exposed to TNT wastes. All stations were sampled on September 23, 1974.

sampled in Grant Creek indicated the most effects, with discharges changing pH, total alkalinity, total solids, sulfates, nitrates, COD, and color. Jerger et al. (1976) sampled Grant Creek and the TNT ditch in Joliet AAP during June 1975 and found several affected water quality parameters (Table 8.36). The values for the TNT ditch are particularly informative and illustrate the severe impact of the wastewater on pH, nitrate levels, conductance, and hardness. The most detailed study at JAAP was reported by Stilwell et al. (1976). They sampled the water and sediments of Grant Creek, Prairie Creek and Doyle Lake in the spring and fall of 1975. The water parameters affected were similar in all three sites (Table 8.37), with increases in solids (suspended and total), and nutrient enrichment (primarily nitrogen species). These effects were matched with a pattern of decreasing TNT concentrations below the pink water discharge (Table 8.38). TNT did accumulate in the sediments, and Doyle Lake sediments also showed increases in COD, nitrogen concentrations, and phosphate concentrations.

Water quality studies were also performed on the Iowa AAP with three reports providing data (Weitzel et al. 1975; Jerger et al. 1976; Sanocki et al. 1976). Weitzel et al. sampled the area streams in August 1974

TABLE 8.32. SUMMARY OF WATER QUALITY DATA IN VACONDA BAY AT VOLUNTEER ARMY AMMUNITION PLANT (VAAP)[a]

Parameters (Units)	Sample Date	Sample Sites										
		A-1	B-1	B-2	C-1	C-2	D-1	E-1	E-1	E-2	T_1[b]	T_2
Distance from VAAP (mile)	-	0.0	0.35	0.35	0.70	0.70	1.0	1.0	1.5	1.5	-	-
Dissolved oxygen (ppm)	6/75	7.08[c]	7.63	6.46	6.69	6.53	7.0	7.0	6.82	7.12	6.49	6.74
Dissolved oxygen (ppm)	8/75	7.10	9.14	6.24	7.36	7.56	7.49	6.36	5.62	7.29	6.87	7.78
Temperature (°C)	6/75	24.0	25.0	24.2	24.4	24.3	24.4	24.2	23.9	24.2	24.4	24.8
Temperature (°C)	8/75	27.0	28.3	26.6	27.6	27.7	27.7	27.0	27.1	27.6	27.0	28.1
pH	6/75	7.5	7.5	7.6	7.7	7.6	7.7	7.8	7.7	7.8	7.7	7.7
pH	8/75	7.7	8.0	7.6	8.0	8.1	8.3	7.7	7.6	7.9	7.9	7.9
Sp. cond. (umhos/cm)[d]	6/75	774	287	388	221	283	185	180	170	161	169	162
Sp. cond. (umhos/cm)	8/75	498	323	412	243	244	205	239	209	176	191	177
TH (mg $CaCO_3$/L)[e]	6/75	125	88.6	97.8	71.6	75.2	69.6	65.6	64.8	66	65.4	66.4
TH (mg $CaCO_3$/L)	8/75	105	91.7	97.7	77.0	79.3	73.0	71.0	70.0	72.7	67.3	67.3
Sulfate (mg SO_4/L)	6/75	108	52.6	80.0	26.6	25.5	25.3	14.7	27.4	16.3	17.4	17.2
Sulfate (mg SO_4/L)	8/75	62.2	50.3	53.0	18.7	23.5	16.9	17.7	14.3	14.1	12.8	12.2
Chloride (mg Cl/L)	6/75	17.4	8.5	12.0	4.8	6.2	3.3	3.9	3.3	2.8	3.4	3.1
Chloride (mg Cl/L)	8/75	41.7	28.5	28.0	11.3	13.5	10.3	9.3	8.3	6.8	6.2	6.7
TDS (mg/L)[f]	6/75	275	183	201	109	127	109	103	94	88	103	103
TDS (mg/L)	8/75	190	232	201	146	193	90	153	158	122	154	140
Total solids (mg/L)	6/75	285	192	211	122	138	117	111	103	96	111	113
Total solids (mg/L)	8/75	198	243	215	159	202	97	160	164	127	161	147
COD (mg/L)[g]	6/75	9.2	8.0	9.0	6.6	7.6	6.9	6.0	6.4	6.1	6.8	5.7
COD (mg/L)	8/75	5.8	11.0	8.6	9.4	7.7	7.0	6.1	5.0	4.9	4.4	4.4
TOC (mg C/L)[h]	6/75	7.1	6.1	6.5	6.1	6.3	5.5	5.8	5.5	5.5	6.1	5.8
TOC (mg C/L)	8/75	4.8	6.1	5.9	8.0	7.1	5.8	5.6	4.9	5.5	6.0	5.9
TKN (mg N/L)[i]	6/75	0.74	0.69	0.76	0.42	0.34	0.36	0.36	0.37	0.34	0.33	0.36
TKN (mg N/L)	8/75	1.88	0.89	0.85	0.51	0.50	0.41	0.36	0.32	0.29	0.32	0.35
Ammonia (mg N/L)	6/75	0.32	0.19	0.17	0.05	0.03	0.03	0.03	0.02	0.02	0.03	0.03
Ammonia (mg N/L)	8/75	1.12	0.34	0.34	0.09	0.08	0.06	0.08	0.08	0.06	0.08	0.07
Nitrite (mg N/L)	6/75	0.251	0.117	0.164	0.038	0.053	0 036	0.028	0.023	0.021	0.020	0.019
Nitrite (mg N/L)	8/75	0.181	0.112	0.115	0.043	0.037	028	0.023	0.017	0.012	0.010	0.010
Nitrate (mg N/L)	6/75	5.04	2.57	3.34	0.92	1.14	0.83	0.63	0.63	0.48	0.54	0.52
Nitrate (mg N/L)	8/75	2.08	1.67	1.68	1.18	1.01	0.83	0.67	0.50	0.41	0.38	0.33
2,4-DNT (ppb)[j]	6/75	68.4	48	66.2	<2	27.2	<2	24.8	<2	0	0	0
2,4-DNT (ppb)	8/75	39.5	16.2	54.2	26.4	14	0	3.8	<2	<2	0	0
2,6-DNT (ppb)[k]	6/75	45	15.8	26.4	13.6	32	0	<2	<2	0	0	0
2,6-DNT (ppb)	8/75	44.8	5.4	80.2	48.4	26.2	0	<2	<2	0	0	0
TNT (ppb)[l]	6/75	24.8	16.8	22.8	2.8	0.8	<2	2.6	4.2	0	0	0
TNT (ppb)	8/75	36.5	7.4	13.6	1.8	<2	0	<2	<2	<2	0	0

a. Adapted from Sullivan et al. 1977.

b. T_1 + T_2 indicate reference samples taken from an adjacent non-TNT-polluted bay.

c. All values are means of 2- determinations.

d. Sp. cond. = specific conductance.

e. TH = total hardness.

f. TDS = total dissolved solids.

g. COD = chemical oxygen demand.

h. TOC = total organic carbon.

i. TKN = total Kjeldhal nitrogen.

j. 2,4-DNT = 2,4-dinitrotoluene.

k. 2,6-DNT = 2,6-dinitrotolene.

l. TNT = 2,4,6-trinitrotoluene.

TABLE 8.33. SUMMARIZED MEAN VALUES FOR SEDIMENT SAMPLES TAKEN IN JUNE AND AUGUST FROM WACONDA BAY, DOWNSTREAM FROM VOLUNTEER ARMY AMMUNITION PLANT[a]

Parameter	Upper Waconda Bay[b]	Lower Waconda Bay[c]	Reference Bay "A"	Huss Lowe Slough
Volatile solids (%)	7.9	4.4	4.6	4.0
Chemical oxygen demand (mg/kg dry wt)	8.9	5.5	4.1	3.2
Total Kjeldahl nitrogen (gm N/kg dry wt)	1.6	1.0	0.7	0.4
Nitrate nitrogen (mg N/kg dry wt)	62	33	54(29[d])	49(27[d])
Total phosphorus (gm P/kg dry wt)	1.07	0.49	0.34	0.17
Trinitrotoluene (mg/kg dry wt)	0.80	<0.1	<0.1	<0.1

a. Adapted from Sullivan et al. 1977.

b. Upper Waconda Bay - A, Transects B and C.

c. Lower Waconda Bay - Transects D, E, F.

d. Excludes two values that appear to be unreasonably high.

and found suggestive changes in water quality parameters in Long Creek and Spring Creek (Table 8.39). However, a similar pattern of effects was not found in Brush Creek, which also bisects IAAP. The significant changes centered on increases in nitrogen species, COD, chloride, and sulfates. Jerger et al. focused on Brush Creek, sampling in June and October of 1975. Table 8.40 shows the affected water quality parameters, with the most notable effects being increased dissolved solids and nutrient enrichment. Sediment analysis in Brush Creek indicated that phosphorus and TKN concentrations were increased, particularly at stations 2 and 4. Munitions and associated compounds were also found at higher concentrations in sediments as shown in Table 8.41. The extremely high values found for site 4 were related to an abandoned "pink water" lagoon that had not been in use for over 20 years. Sanocki et al. (1976) continued the study by Jerger et al. and provided new data on Spring Creek and several point and nonpoint AAP sources to Brush Creek, which they called industrial stations. They sampled these areas in June and October of 1975. The data on Spring Creek did not reveal any significant differences between the two sampling sites; however, the samples from industrial stations did indicate that many of the changes found in Brush Creek (reported by Jerger et al. 1976) were related to IAAP effluents. As shown in Table 8.42, station 1 contributed the

TABLE 8.34. WATER ANALYSES OF THE IMPACT OF HOLSTON ARMY AMMUNITION PLANT (HAAP) ON HOLSTON RIVER[a]

Parameters	Stations[b]					
	A	A_1	Je	Jf	K_2	M
Alkalinity (ppm $CaCO_3$)	60	70	77	60	72	105
Color (CP units)	7	10	12	23	18	23
Sulfates (ppm SO_4)	11	13	22	33	21	32
Total solids (ppm)	96	109	199	170	189	196
Total dissolved solids (ppm)	96	109	198	164	189	187
Total suspended solids (ppm)	<1	<1	1	6	<1	9
Total volatile solids (ppm)	69	52	48	49	71	40
Nitrates (ppm N)	1.00	1.14	0.04	0.09	2.14	0.01
Nitrites (ppm N)	<0.05	<0.05	<0.05	<0.05	1.16	<0.05
Total Kjeldahl nitrogen (ppm N)	0.70	1.37	1.33	1.80	1.42	1.19
Ammonia (ppm)	<0.25	<0.25	<0.25	<0.25	<0.25	<0.25
Orthophosphates (ppm P)	<0.01	0.05	0.02	0.08	<0.01	0.01
Chemical oxygen demand (ppm)	18.0	22.5	40.5	36.0	49.5	607.0
Total organic carbon (ppm)	7	6	14	23	16	120
Trinitrotoluene (ppm)	<0.1	<0.1	<0.1	<0.1	<0.1	<0.1
Trinitrobenzene (ppm)	<0.2	<0.2	<0.2	<0.2	<0.2	<0.2
2,4-Dinitrotoluene (ppm)	<0.1	<0.1	<0.1	<0.1	<0.1	<0.1
2,6-Dinitrotoluene (ppm)	<0.1	<0.1	<0.1	<0.1	<0.1	<0.1

a. Adapted from Huff et al. 1975c.

b. Stations A, A_1, and K_2 were sampled on September 24; Stations J and M were sampled on September 26. Stations A and A_1 are upstream of HAAP, but below industrial discharge points; stations Je and Jf [Je and Jf = J(effluent side) and J(far side), respectively] are located below explosive production lines; station K_2 is below magazine area, and station M is at the downstream boundary of HAAP.

TABLE 8.35. WATER QUALITY DATA FOR THE JOLIET ARMY AMMUNITION PLANT-ASSOCIATED STREAMS[a]

Parameters[b]	Grant Creek		Jackson Creek		Prairie Creek	
	Downstream	Upstream	Downstream	Upstream	Downstream	Upstream
pH, Units	6.5	7.6	7.2	7.4	7.3	7.3
COD	178	20	108	108	163	178
BOD_5	<10					
Total alkalinity, as $CaCO_3$	21.3	221	96	114.5	98.5	94.5
Total acidity, as $CaCO_3$		12.6	13.5	7.25	8.9	7.6
Nitrates, as N	21.4	5.23			19.76	18.86
Total nitrogen, as N		7.55	18.87	19.03	29.02	25.81
Sulfates	281		173	214	149	127
Suspended solids	96	248	1200	704	1150	1400
Volatile suspended solids	19	35	151		127	202
Total solids	978	664	1530	1030	1637	1750
Total volatile solids	198	328	323	208	321	427
Dissolved solids	882	416	830	326	478	350
Settleable solids, ml/L	0.08	1.1	1.88	1.33	1.87	3.11
Chlorides	23.3	3.3	8.93	0		4.7
Organic carbon	14.2	21	69	67	79	130
Total carbon	16.5	76	94	90	111	145
Color, PCU	150	20	40	50		40

a. Data taken from Rychman, Edgerley, Burbank, and Associates, Report on Waste-water Control and Treatment Program for Uniroyal, Inc. June 1968, as cited in Ghassemi et al. 1976.

b. Except as noted, all values in mg/L.

TABLE 8.36. SUMMARY OF WATER QUALITY DATA AT JOLIET ARMY AMMUNITION PLANT[a]

Parameter[b] (Units)	Grant Creek Upstream Site	Grant Creek Downstream Site	TNT Ditch Upstream Site	TNT Ditch Downstream Site
Sp. cond. (μmhos/cm)	710	800	720	3,550
Tot. sol. (mg/L)	502	563	492	789
pH	8.18	7.90	7.90	2.51
Tot. alk. (mg/L as $CaCO_3$)	228	211	151	0
Sulfate (mg/L)	94	120	120	250
Tot. hard. (mg/L as $CaCO_3$)	312	319	225	664
Calcium (mg/L)	72.2	72	47.3	141
Sodium (mg/L)	12.8	24.2	48.3	120
COD (mg/L)	<5	11	15	49
TOC (mg/L)	14	11	10	17
TKN (mg/L)	0.3	0.5	1.9	0.1
Ammonia-N (mg/L)	0.096	0.37	1.4	1.2
Nitrite-N (mg/L)	0.069	0.18	0.58	0.52
Nitrate-N (mg/L)	8.4	13	15	260
Tot. P (mg/L)	0.11	0.093	0.090	0.14
2,6-DNT (μg/L)	<0.1	51	380	3,550
2,4-DNT (μg/L)	<0.1	320	1,370	7,380
2,4,6-TNT (μg/L)	<0.2	86	430	1,020

a. Adapted from Jerger et al. 1976.

b. Sp. cond. = specific conductance; Tot. sol. = total solids; Tot. alk. = total alkalinity; Tot. hard. = total hardness; COD = chemical oxygen demand; TOC = total organic carbon; TKN = total kjeldahl nitrogen; Tot. P = total phosphorus; 2,6-DNT = 2,6-dinitrotoluene; 2,4-DNT = 2,4-dinitrotoluene; 2,4,6-TNT = 2,4,6-trinitrotoluene.

majority of pollutants and is described by Sanocki et al. as a nonmunitions source. The other sources did contribute munition-related pollutants, with most of the pollution being nitrogen and phosphorus enrichments. Also shown in Table 8.42 are the levels of munition compounds in the water phase of the various effluents. These data indicate that industrial stations do contribute detectable amounts of the munition compounds.

Keirn et al. (1981) reported the water quality and TNT levels for the Alabama AAP, which produced TNT for three years in the 1940s. They sampled the three main drainage systems of the plant, which they called the Crossover Ditch, the Red Water Ditch, and the Beaver Pond. Table 8.43 shows the water quality data for the drainages and indicates that the contamination levels were low. The majority of effects were in drainages associated with the TNT-manufacturing areas. The Crossover Ditch had very high sulfate and specific conductance values at some stations. However, the Red Water Ditch showed the most overall effects of any system, and this trend was further supported by munition levels found in the sediments (Table 8.44). These levels were very high, especially for TNT, and indicate a potential for extended impact on the aquatic system.

TABLE 8.37. WATER QUALITY DATA FOR TNT DISCHARGES FROM JOLIET ARMY AMMUNITION PLANT TO ASSOCIATED SURFACE WATERS[a]

	Parameters[b]													
	Conductance	Temperature (°C)	Alkalinity	Total Hardness	Suspended Solids	Dissolved Solids	COD[c]	TOC[d]	TKN[e]	NH_4	NO_2	NO_3	PO_4	SO_4	Cl
Grant Creek															
Spring															
Upstream control	6.3	18.0	227	308	232	476	35	7.7	1.9	0.20	0.34	43.7	0.17	73	22
Below filtration plant outfall	1115	19.7	287	412	394	999	36	9.6	2.0	0.11	0.41	133.4	0.06	290	37
Red water ditch	995	28.7	170	359	29	638	29	48	2.1	0.26	3.6	53	0.05	191	24
Downstream	865	21.5	249	368	258	627	34	8.4	1.5	0.19	1.04	76.2	0.07	145	28
Fall															
Upstream control	705	14.3	289	356	48	482	6	8.5	1.2	0.13	0.03	3.0	0.13	77	19
Red water ditch outfall	425	51	64	19	15	181	154	52	22	42	74		0.05	36	3.8
Downstream	1118	21.7	211	452	95	914	19.5	13.8	1.0	0.74	2.91	95.0	0.14	187	95
Doyle Lake															
Spring															
Discharge to Doyle Lake	1272	20.5	250	204		773	18	6.7	0.97	<0.18	0.13	13.3	0.08	192	122
Doyle Lake	596	23.4	191	249	46	385	32	7.7	1.5		0.33	75	0.06	59	19.5
Fall															
Discharge to Doyle Lake	927	17.3	268	263	55	655	12	6	0.45	0.13	0.06	4.0	0.37	121	99
Doyle Lake	946	18.4	253	214	47	633	43	15	1.3	0.08			0.17	131	95
Prairie Creek															
Fall															
Upstream	795	16.6	274	419	29	602	10	5.0	0.5	0.07			0.26	162	8
Downstream	973	18.0	265	300	23	619	13	7.5	0.3	0.07	0.04	1.3	0.35	166	62

a. Adapted from Stilwell et al. 1976.

b. Values are means given in ppm, unless indicated otherwise.

c. COD = chemical oxygen demand.

d. TOC = total organic carbon

e. TKN = total Kjeldahl nitrogen.

TABLE 8.38. CONCENTRATIONS OF TRINITROTOLUENE (TNT), 2,4-DINITROTOLUENE (2,4-DNT), AND 2,6-DINITROTOLUENE (2,6-DNT) IN SELECTED WATER SAMPLES FROM JOLIET ARMY AMMUNITION PLANT-AFFECTED SITES[a]

Sites[b]	TNT[c] (ppb)	2,4-DNT[d] (ppb)	2,6-DNT[e] (ppb)
	Grant Creek		
	Spring 1975		
Red water ditch			
RW-0	75 (4.2-145.7)[f]	258 (7.2-509.7)	85 (2.9-167.3)
RW-S	55 (24.8-133.2)	16 (12.4-21.2)	24 (12.1-62.7)
Downstream			
GC-4	25 (7.6-63.7)	12 (2.2-43.6)	28 (4.6-49.5)
GC-5	9 (4.0-12.4)	10 (6.0-15.9)	13 (13.0-14.0)
	Fall 1975		
Red water ditch			
RW-0	23 (19.4-24.8)	63 (55.0-67.7)	20 (18.1-22.1)
Downstream			
GC-4	30(26.7-32.5)	19 (18.9-19.5)	15 (13.5-15.8)
GC-5	28 (18.0-37.7)	17 (11.8-21.8)	15 (12.6-16.8)
	Doyle Lake		
	Spring 1975		
Discharge			
DL-0	24 (7.0-31.0)	1.1 (<0.2-2.7)	0.8 (0.6-0.9)
DL-1	28 (3.5-65.5)	0.3 (<0.2-0.3)	0.7 (<0.2-1.3)
Doyle Lake			
DL-2	<0.6	0.2 (<0.2-0.3)	0.6 (<0.4-1.0)
DL-3	<0.6	0.2 (<0.2-0.2)	0.8 (<0.4-1.2)
Outflow			
DL-4	<0.6	1.8 (<0.2-4.1)	0.9 (<0.4-1.4)
	Fall 1975		
Discharge			
DL-0	40.1	<0.2	<0.4
DL-1	43 (2.8-83.6)	6.5 (3.2-9.7)	<0.4
Doyle Lake			
DL-2	<0.6	<0.5 (0.4-0.6)	<0.4
DL-3	<0.6	<0.2	<0.4
Outflow			
DL-4	<0.6	0.3 (<0.2-0.3)	<0.4
	Prairie Creek		
	Fall 1975		
Discharge			
PC-0	35 (33.2-36.0)	<0.2	<0.4
Prairie Creek			
PC-3	<10 (<0.6-6.4)	<0.2	<0.4
PC-4	<10 (<0.6-7.5)	<0.2	<0.4

a. Adapted from Stilwell et al. 1976.

b. RW-0 = red water treatment plant outflow; RW-S = redwater treatment plant discharge stream to Grant Creek; GC-4 = Grant Creek downstream from pink water effluent; GC-5 = Grant Creek at plant boundary; DL-0 = LAP line effluentd; DL-1 = Doyle Lake effluent stream; DL-2 + DL-3 = Doyle Lake settling pond; DL-4 = Doyle Lake outlet; PC-0 = LAP line effluent outfall to Prairie Creek; PC-3 + PC-4 = Prairie Creek below LAP line outfall.

c. TNT = 2,4,6-trinitrotoluene.

d. 2,4-DNT = 2,4-dinitrotoluene.

e. 2,6-DNT = 2,6-dinitrotoluene.

f. Values are means and ranges.

TABLE 8.39. SUMMARY OF WATER QUALITY DATA AT IOWA ARMY AMMUNITION PLANT (IAAP)[a] - I

Parameter[b] (Units)	Long Creek1[c]	Long Creek2[d]	Long Creek3[e]	Long Creek4[f]	Spring Creek1[c]	Spring Creek2[d]	Spring Creek3[g]
TSS (mg/L)	9	22	4	6	17	10	12
Tot. Hard. (mg/L[h])	173	214	157	169	270	299	255
Chloride (mg/L)	15	20	12	11	27	182	53
Sulfate (mg/L)	39	43	38	43	43	206	122
BOD (mg/L)	0.9	0.9	1.5	1.3	1.5	5.1	1.2
COD (mg/L)	6	12	58	13	11	23	9
Nitrite-N (mg/L)	0.011	0.075	0.094	0.009	0.032	0.910	0.090
Nitrate-N (mg/L)	1.2	4.7	1.9	1.1	0.75	5.8	3.8
TKN (mg/L)	0.1	2.0	3.1	1.6	4.9	10.1	2.8
Tot. P (mg/L)	0.20	0.008	0.001	0.003	0.012	0.194	0.049
2,4-DNT (µg/L)	<0.1[i]	<0.1	0.3	<0.1	0.3	0.4	<0.1
2,4,6-TNT (µg/L)	<0.2	0.2	3.0	<0.2	1.0	3.4	<0.2

a. Adapted from Weitzel et al. 1975.

b. TSS = total suspended solids; Tot. hard. = total hardness; COD = chemical oxygen demand; TKN = total kjeldahl nitrogen; Tot. P = total phosphorus; 2,4-DNT = 2,4-dinitrotoluene; 2,4,6-TNT = 2,4,6-trinitrotoluene.

c. Sample taken at upstream control.

d. Sample taken downstream from treatment effluent.

e. Sample taken in reservoir near dam.

f. Sample taken downstream from reservoir

g. Sample taken at downstream boundary of IAAP.

h. Measured as $CaCO_3$

i. < Values indicate the sample was below limits of detection.

TABLE 8.40. SUMMARY OF WATER QUALITY DATA AT IOWA ARMY AMMUNITION PLANT (IAAP)[a] - II

Parameter[b] (Units)	Brush Creek1[c]	Brush Creek2[d]	Brush Creek3[e]	Brush Creek4[f]	Brush Creek5[d]	Brush Creek6[g]	Brush Creek7[h]	Brush Creek8[i]
June 1975[j]								
Tot. Sol. (mg/L)	355	476	467	412	382	566	428	365
Chloride (mg/L)	37.1	109	97.3	72.8	58.6	162	110	68.8
Sulfate (mg/L)	38	73	71	71	61	61	61	60
Tot. Hard. (mg/L[k])	280	159	164	151	149	194	181	185
Sodium (mg/L)	12	80	99	84	61	105	70	45
COD (mg/L)	<8[l]	14	14	23	18	10	15	10
TOC (mg/L)	4	15	25	16	10	12	9	,
TKN (mg/L)	0.8	0.7	0.7	0.8	2.0	0.6	0.9	0.5
Ammonia-N (mg/L)	0.084	0.083	0.11	0.12	1.5	0.11	0.42	0.075
Nitrite-N (mg/L)	0.028	0.009	0.010	0.016	0.11	0.010	0.008	0.005
Nitrate-N (mg/L)	3.6	4.1	3.7	3.2	4.3	2.4	4.6	4.1
Tot. P (mg/L)	0.073	0.58	0.56	0.52	0.47	0.59	0.94	0.72
2,4-DNT (µg/L)	<0.2	<0.1	<0.1	<0.2	0.1	<0.1	<0.1	0.1
2,4,6-TNT (µg/L)	<0.2	<0.2	<0.2	2.5	3.4	0.3	4.1	1.3
1,3,5-TNT (µg/L)	<0.6	0.2	<0.2	<0.2	0.4	<0.2	0.4	0.7
October 1975								
Tot. Sol. (mg/L)	1480	789	776	724	854	711	469	486
Chloride (mg/L)	25.7	241	234	258	353	209	109	136
Sulfate (mg/L)	870	98	100	100	96	96	67	77
Tot. Hard. (mg/L[k])	890	229	213	204	236	205	185	223
Sodium (mg/L)	34	172	176	170	187	142	75	86
COD (mg/L)	14	18	19	20	17	21	11	14
TOC (mg/L)	6	7	8	6	7	6	6	5
TKN (mg/L)	0.8	0.9	0.8	0.8	0.7	0.7	0.9	0.7
Ammonia-N (mg/L)	0.44	0.24	0.28	0.22	0.15	0.043	0.086	0.032
Nitrite-N (mg/L)	0.003	0.005	0.007	0.005	0.008	0.007	0.004	0.007
Nitrate-N (mg/L)	0.092	0.40	0.30	0.31	0.45	1.1	3.1	1.5
Tot. P (mg/L)	0.030	0.71	0.77	0.75	1.6	3.7	2.2	1.6
2,4-DNT (µg/L)	<0.1	0.1	0.1	0.1	<0.1	<0.1	0.1	<0.1
2,4,6-TNT (µg/L)	<0.2	0.5	0.2	0.8	0.5	<0.2	0.5	0.3
1,3,5-TNT (µg/L)	<0.2	0.6	0.3	0.5	<0.2	<0.2	<0.2	<0.2

a. Adapted from Jerger et al. 1976.

b. Tot. sol. = total solids; Tot. hard. = total hardness; COD = chemical oxygen demand; TOC = total organic carbon; TKN = total Kjeldahl nitrogen; Tot. P = total phosphorus; 2,4-DNT = 2,4-dinitrotoluene; 2,4,6-TNT = 2,4,6-trinitrotoluene; 1,3,5-TNT = 1,3,5-trinitrotoluene.

c. Sample taken at upstream control.

d. Sample taken downstream from munition manufacturing effluent.

e. Sample taken downstream from process effluent.

f. Sample taken downsteam from historic munition dump.

g. Sample taken above sewage treatment plant.

h. Sample taken downstream from treatment plant.

i. Sample taken downstream at boundary of IAAP.

j. Indicates the period when the sample was taken.

k. Measured as $CaCO_3$.

l. < Values indicate the sample was below limits of detection.

TABLE 8.41. SEDIMENT PHASE MUNITIONS DATA, IOWA ARMY AMMUNITION PLANT[a]

Parameter (mg/kg)	Brush Creek Stations[b]							
	B1	B2	B3	B4	B5	B6	B7	B8
June 1975								
2,6-Dinitrotoluene	<0.1	2.4	<0.1	<0.1	<0.1	<0.1	0.1	<0.1
2,4-Dinitrotoluene	<0.4	<0.1	<0.1	0.1	<0.1	0.1	0.2	<0.1
1,3,5-Trinitrobenzene	<1.3	3.5	<1.0	1.4	<1.0	1.2	<1.0	<1.0
2,4,6-Trinitrotoluene	<1.0	9.0	<0.2	18.7	0.3	1.0	2.7	<0.2
4-Hydroxylamino-2,6-dinitrotoluene	<5	8	<5	9	<5	<5	<5	<5
2-Hydroxylamino-4,6-dinitrotoluene	<30	90	<30	45	<30	<30	<30	<30
October 1975								
2,6-Dinitrotoluene	<0.2	0.3	<0.1	0.1	<0.1	<0.1	<0.1	<0.1
2,4-Dinitrotoluene	<0.3	<0.1	<0.1	0.9	0.1	0.3	0.1	<0.1
1,3,5-Trinitrobenzene	<3.7	1.6	<1.0	2.0	<1.0	5.1	<1.0	1.3
2,4,6-Trinitrotoluene	<0.2	2.6	<0.2	111	2.0	2.0	0.3	0.4
4-Hydroxylamino-2,6-dinitrotoluene	<5	<5	<5	57	<5	<5	<5	<5
2-Hydroxylamino-4,6-dinitrotoluene	<43	44	<30	101	33	<30	<30	<30

a. Adapted from Jerger et al. 1976.

b. Values are means of 0-10-cm section samples; stations are described in footnote to Table 8.40.

In summary, the effects of TNT on water quality are concentrated on several parameters. TNT wastewater was found to increase the COD, the load of solids (both dissolved and suspended), the levels of nitrogen species (nitrates, nitrites, and TKN), and levels of sulfates of the water in receiving streams at a majority of AAP locations. Affected to a lesser degree were TOC, pH, and phosphate levels. The effects on associated sediments paralleled those found for the water column, with the greatest impact on COD, levels of nitrogen species, and phosphate concentrations. The expected significance of these effects would be to increase nutrients for algal and plant growth, reduce light penetration and substrate habitat availability due to input of solids, reduce oxygen availablity for animal utilization and organic decomposition, and, in general, increase the frequency of changes in water parameters (e.g., in pH) that are stressful to the biotic communities.

8.4.1.2 HMX, RDX, and Associated Wastes

Little information was found in the literature for the effects of released RDX or HMX processing effluents on the water quality of natural systems. The majority of data were measurements of water quality parameters of load, assemble, and pack (LAP) effluents prior to release from the facilities. These data should at least indicate the potential for

TABLE 8.42. SUMMARY OF WATER QUALITY DATA AT IOWA ARMY AMMUNITION PLANT (IAAP)[a] – III

Parameter[b] (Units)	Industrial St1[c]	Industrial St2[d]	Industrial St3[e]	Industrial St4[f]	Industrial St5[g]	Industrial St6[h]	Industrial St7[h]
June 1975[i]							
Sp. Cond. (μmhos/cm)	8,290	440	411	303	348	NA[j]	352
Tot. Sol. (mg/L)	6,380	299	275	179	238	NA	225
TSS (mg/L)	130	9	4	3	15	NA	11
pH	10.95	7.75	7.95	8.30	7.65	NA	7.75
Tot. Alk. (mg/L[k])	327	118	126	124	91	NA	98
Chloride (mg/L)	2,620	37.5	27.5	16.2	30.1	NA	30.9
Sulfate (mg/L)	215	51	45	21	39	NA	38
Tot. Hard. (mg/L[k])	780	180	181	154	145	NA	160
Sodium (mg/L)	1,560	29	24	12	23	NA	22
BOD (mg/L)	15	2	2	1	2	NA	5
COD (mg/L)	57	13	7	39	16	NA	32
TOC (mg/L)	24	8	15	5	7	NA	5
TKN (mg/L)	1.2	0.7	0.6	0.4	0.8	NA	5.1
Ammonia-N (mg/L)	0.075	0.076	0.080	0.075	0.083	NA	4.1
Nitrite-N (mg/L)	0.009	0.005	0.003	0.004	0.005	NA	0.15
Nitrate-N (mg/L)	14	2.7	2.0	2.5	2.5	NA	8.0
Tot. P (mg/L)	5.4	0.16	0.074	0.091	0.82	NA	0.048
2,4-DNT (μg/L)	<0.1[l]	<0.1	0.2	<0.1	0.2	NA	<0.1
2,4,6-TNT (μg/L)	<0.2	<0.2	0.5	11.7	0.4	NA	3.4
4-HA-2,6-DNT (μg/L)	<6	<5	6	5	23	NA	7
2-HA-4,6-DNT (μg/L)	<10	<10	10	10	32	NA	11
October 1975							
Sp. Cond. (μmhos/cm)	2,180	362	388	292	376	NA	358
Tot. Sol. (mg/L)	1,200	224	271	197	321	NA	230
TSS (mg/L)	172	8	2	6	10	NA	5
pH	10.85	7.90	7.80	7.90	6.70	NA	7.95
Tot. Alk. (mg/L[k])	411	114	115	100	72	NA	115
Chloride (mg/L)	337	43.5	59.6	36.9	39.6	NA	42.9
Sulfate (mg/L)	240	45	60	36	52	NA	51
Tot. Hard. (mg/L[k])	35	160	170	136	145	NA	166
Sodium (mg/L)	369	37	39	32	48	NA	35
BOD (mg/L)	20	1	3	1	1	NA	1
COD (mg/L)	74	8	12	12	9	NA	6
TOC (mg/L)	16	4	6	4	6	NA	4
TKN (mg/L)	1.3	0.4	1.1	0.5	0.6	NA	0.4
Ammonia-N (mg/L)	0.062	0.050	0.60	0.068	0.18	NA	0.083
Nitrite-N (mg/L)	0.001	0.003	0.017	0.005	0.003	NA	0.057
Nitrate-N (mg/L)	0.67	0.11	0.49	0.38	5.2	NA	0.56
Tot. P (mg/L)	8.2	0.11	0.071	1.1	32	NA	0.042
2,4-DNT (μg/L)	<0.1	<0.1	<0.1	0.2	<0.1	NA	<0.1
2,4,6-TNT (μg/L)	<0.2	<0.2	<0.2	16.7	<0.2	NA	6.0
4-HA-2,6-DNT (μg/L)	<5	<5	7	5	<5	NA	11
2-HA-4,6-DNT (μg/L)	<10	<10	14	14	<10	NA	20

a. Adapted from Sanocki et al. 1976.

b. Sp. cond. = specific conductance; Tot. sol. = total solids; TSS = total suspended solids; Tot. alk. = total alkalinity; Tot. hard. = total hardness; BOD = biological oxygen demand; COD = chemical oxygen demand; TOC = total organic carbon; TKN = total kjeldahl nitrogen; Tot. P = total phosphorus; 2,4-DNT = 2,4-dinitrotoluene; 2,4,6-TNT = 2,4,6-trinitrotoluene; 4-HA-2,6-DNT = 4-hydroxyamino-2,6-dinitrotoluene; 2-HA-4,6-DNT = 2-hydroxyamino-4,6-dinitrotoluene.

c. Sample at industrial station with boiler blowdown effluent.

d. Sample at industrial station with munition effluent.

e. Sample at industrial station with TNT melt effluent.

f. Sample at industrial station with TNT process effluent.

g. Sample at industrial station with metal refinish effluent.

h. Sample at industrial station with carbon contact effluent.

i. Indicates the period when the sample was taken.

j. NA = not available.

k. Measured as $CaCO_3$.

l. < Values indicate the sample was below limits of detection.

TABLE 8.43. SUMMARY OF WATER QUALITY DATA AT ALABAMA ARMY AMMUNITION PLANT (AAAP)[a]

Sample Location[f]	Flow Status[g]	Sp. Cond.[b] (umho/cm)	pH	Nitrate/Nitrite (ppm as N)	Sulfate (ppm)	2,4-DNT[c] (ppb)	2,6-DNT[d] (ppb)	2,4,6-TNT[e] (ppb)
Crossover Ditch Drainage								
LBA	F	5,570	2.5	0.322	14,800	<1.2[h]	<1.3	<0.41
LBA	P	486	3.5	0.236	241	<1.2	<1.3	<0.41
LBA/RPF	F	388	4.9	<0.100	225	<1.2	<1.3	<0.41
RPF	F	88	5.8	<0.100	<7	<1.2	<1.3	<0.41
PS	P	55	5.5	<0.100	<7	<1.2	<1.3	<0.41
BT	P	70	5.7	<0.100	<7	<1.2	<1.3	<0.41
S.TNT	P	115	6.6	<0.100	10	<1.2	<1.3	<0.41
B/SL	P	113	6.1	0.101	<7	<1.2	<1.3	<0.41
ALL	P	184	6.4	<0.100	54	<1.2	<1.3	<0.41
B/SF	F	162	6.2	0.587	<37	<1.2	<1.3	<0.41
ALL	F	190	6.4	<0.100	<74	<1.2	<1.3	0.41
Red Water Ditch Drainage								
AM/TM	P	350	3.9	0.132	193	<1.2	<1.3	<0.41
AM/TM	F	139	5.2	<0.100	42	ND[i]	ND	ND
AM	P	115	5.6	0.334	19	<1.2	<1.3	<0.41
ALL	P	440	6.0	5.69	210	3.7	7.1	9.3
N.TNT	P	444	6.2	<5.0	220	3.4	4.7	5.5
ALL	P	98	6.5	<0.100	26	3.0	2.6	13.9
ALL	F	185	6.3	<0.100	42	<1.2	<1.3	<0.41
Beaver Pond Drainage								
N.TNT	F	207	6.6	0.451	23	<1.2	<1.3	140
PS	P	70	5.2	<0.100	8	<1.2	<1.3	<0.41
ALL	P	156	6.8	0.324	8	<1.2	<1.3	<0.41
Misc. Drainages								
RWSB	P	81	6.2	<0.100	<7	<1.2	<1.3	<0.41
FG	P	92	5.3	<0.100	10	<1.2	<1.3	<0.41

a. Adapted from Keirn et al. 1981.

b. Sp. Cond. = specific conductance.

c. 2,4-DNT = 2,4-dinitrotoluene.

d. 2,6-DNT = 2,6-dinitrotoluene.

e. 2,4,6-TNT = 2,4,6-trinitrotoluene.

f. Area of AAAP drained, LBA = leaseback area; RPF = rifle powder finishing; PS = propellant shipping; BT = blending tower; S.TNT = southern TNT manufacturing; B/SF = burn area/sanitary landfill; ALL = includes input from all areas in drainage; AM = acid manufacturing; AM/TM = acid manufacturing/tetryl manufacturing; N.TNT = northern TNT manufacturing; RWSB = red water sludge basin; FG = flashing ground.

g. Conditions at time of sampling, P = pooled; F = flowing.

h. < Values represent the limit of detection for that munition; some detection limits increased due to interference.

i. ND = not determined.

TABLE 8.44. SUMMARY OF SEDIMENT MUNITION LEVELS IN RED WATER DITCH AT ALABAMA ARMY AMMUNITION PLANT (AAAP)[a]

Sample Location[f]	Sulfate (ppm)	2,4-DNT[b] (ppb)	2,6-DNT[c] (ppb)	TNT[d] (ppb)	TNB[e] (ppb)
Acid manufacturing	1,430	<66[g]	<73	<73	<359
Acid/Tetryl manufacturing	173	222	<73	77	<359
Acid manufacturing/ aniline sludge basin	<54	<66	<73	86	<359
All[h]	1,600	60	268	50	<359
All	364	<66	<73	17,600	<359
All	<54	<66	310	3,300	<359
All	175	5,680	1,850	13,900	<359
All	<54	<66	<73	52	<359
All	<54	<66	<73	188	<359
All	<54	<66	<73	104	<359
All	107	<66	<73	<73	<359
All	612	<66	<73	<73	<359
All	464	<66	<73	<73	<359

a. Adapted from Keirn et al. 1981.

b. 2,4-DNT = 2,4-dinitrotoluene.

c. 2,6-DNT = 2,6-dinitrotoluene.

d. TNT = 2,4,6-trinitrotoluene.

e. TNB = 1,3,5-trinitrobenzene.

f. Area of AAAP drained.

g. < Values represent the limit of detection for that substance.

h. All includes drainage input from manufacturing areas for acids, TNT, and Tetryl and input from the aniline sludge basin.

effects on environmental water quality. One report on an AAP did provide data on RDX levels in surface waters and stream sediments (Envirodyne Engineers, Inc., 1980).

The surface water systems of the Milan AAP were evaluated for munition levels by Envirodyne Engineers, Inc. (1980). They found detectable levels of RDX and some by-products (as well as TNT) in waters and sediments of streams traversing the AAP (Table 8.45). Similar patterns of munition concentrations were also found in waste storage lagoons (Table 8.46). The levels of RDX were usually the highest of the munition levels and were often accompanied by high nitrate and sulfate levels. The relative impact of these elevated values is hard to determine since no background values were reported.

TABLE 8.45. SUMMARY OF STREAM WATER QUALITY DATA AT MILAN ARMY AMMUNITION PLANT[a]

Parameter[b]	Total Samples	Number of Positive Samples	Range	Average[c]	Detection Limits
Water Column (µg/L)					
Nitrate	22	22	<100-1,800	359	<100
Nitrite	22	22	<10-20	10.5	<10
Phosphate	22	22	<200-900	291	<200
Sulfate	22	22	3,000-39,000	10,690	NG[d]
2,4-DNT	22	7	<0.006-0.25	0.125	<0.006
2,6-DNT	22	21	<0.001-0.057	0.0023	<0.001
2,4,6-TNT	22	20	0.04-41	2.41	<0.06
RDX	22	13	<0.4-110	14.6	<0.4
Sediments (µg/g)					
Nitrate	15	15	<0.25-0.78	0.32	<0.25
Nitrite	15	15	<0.03-0.10	0.05	<0.03
Phosphate	15	15	<0.5-2.4	0.6	<0.5
Sulfate	15	15	<12-50	21	<12
2,4-DNT	15	3	<6-190	67.3	<3
2,6-DNT	15	10	<0.5-19	4.65	<0.5
2,4,6-TNT	15	15	9-1,200	416	NG
RDX	15	3	290-43,000	15,160	<220

a. Adapted from Envirodyne Engineers, Inc. 1980.

b. 2,4-DNT = 2,4-dinitrotoluene; 2,6-DNT = 2,6-dinitrotoluene; 2,4,6-TNT = 2,4,6-trinitrotoluene; RDX = hexahydro-1,3,5-trinitro-1,3,5-triazine.

c. Average is calculated with < values assumed to be equal to the value itself.

d. NG = not given.

TABLE 8.46. SUMMARY OF LAGOON WATER QUALITY DATA AT MILAN ARMY AMMUNITION PLANT[a]

Parameter[b]	Total Samples	Number of Positive Samples	Range	Average[c]	Detection Limits
Water Column (µg/L)					
Nitrate	5	5	<100-2,900	1,100	<100
Nitrite	5	5	10-90	34	NG[d]
Phosphate	5	5	<200-300	240	<200
Sulfate	5	5	<4,600		<4,600
2,4-DNT	5	2	<20-20	20	<0.06
2,6-DNT	5	3	<0.01-<4	1.98	<0.01
2,4,6-TNT	5	5	0.4-49	23	NG
RDX	5	4	<4-<1,600	400	<320
Sediments (µg/g)					
Nitrate	5	5	<0.25-10	7.4	<0.25
Nitrite	5	5	0.08-12	2.5	NG
Phosphate	5	5	<0.5-210	42	<0.5
Sulfate	5	5	22-73	37	NG
2,4-DNT	5	5	81-330	190	NG
2,6-DNT	5	5	7-59	32	NG
2,4,6-TNT	5	5	2,900-26,000	15,240	NG
RDX	5	5	2,600-38,000	11,080	NG

a. Adapted from Envirodyne Engineers, Inc. 1980.

b. 2,4-DNT = 2,4-dinitrotoluene; 2,6-DNT = 2,6-dinitrotoluene; 2,4,6-TNT = 2,4,6-trinitrotoluene; RDX = hexahydro-1,3,5-trinitro-1,3,5-triazine.

c. Average is calculated with < values assumed to be equal to the value itself.

d. NG = not given.

Stilwell et al. (1977) tested the waste effluents from two manufacturing areas of Holston AAP for munition content and water quality (Table 8.47). Area A contained ten sources of wastewater, with the primary inputs being effluents from acetic acid and anhydride processes and steam boilers. Area B contained four sources of effluents from the production lines and miscellaneous areas. The effluents from both areas contained high amounts of nitrogen species, phosphates, solids, and smaller levels of munition compounds. Biological and chemical oxygen demands were also elevated. Of course the impact of these wastes on the water quality of the Holston River would be moderated by treatment processes and dilution. In a similar analysis of Joliet AAP wastewaters, the treatment processes reduced the impact of most pollutants except total solids (Table 8.48). Spanggord et al. (1978) present data on pre- and posttreatment effluents for Iowa AAP which show a parallel trend (Table 8.49). In this case the pretreatment effluent did not contain extremely high levels of nitrogen species and the posttreatment effluent was even less of a potential hazard.

TABLE 8.47. SUMMARY OF WASTEWATER QUALITY DATA AT HOLSTON ARMY AMMUNITION PLANTa

Parameter (units)	Area Ab	Area Bc	Holston River (Control)
Chemical oxygen demand (ppm)	4,058	314	58
Biological oxygen demand (ppm)	2,798	191	NDd
pH	4.3	7.3	ND
Ammonia-N (ppm)	102.3	13.3	ND
Total Kjeldahl nitrogen (ppm)	126.8	22.7	ND
Phosphates (ppm)	420	24	ND
Nitrates (ppm)	38.9	43.9	ND
Nitrites (ppm)	40	1	ND
Total solids (ppm)	2002	426	ND
HMXe (ppm)	3.59	0.77	<0.05
RDXf (ppm)	0.88	0.80	<0.05
TNTg (ppm)	<0.05	<0.05	<0.05

a. Adapted from Stilwell et al. 1977.

b. Area A effluent is composed of ten wastewater streams from acetic acid concentrators, steam generators, and filter plants.

c. Area B effluent is composed of four wastewater streams from the composition B production lines and the continuous RDX production line.

d. ND = not determined.

e. HMX = 1,3,5,7-tetrahydro-1,3,5,7-tetracyclooctane.

f. RDX = hexahydro-1,3,5-trinitro-1,3,5-triazine.

g. TNT = 2,4,6-trinitrotoluene.

TABLE 8.48. PERFORMANCE OF LAP[a] WASTEWATER TREATMENT AT JOLIET ARMY AMMUNITION PLANT[b]

Parameter[c]	LAP Influent	Effluent Source			Average Percent Change
		Diat.[d] Earth Filter	1st Carbon Column	2nd Carbon Column	
pH	7.9	7.9	7.8	7.7	-
Total solids	1401.5[e]	1418.5	1138.3	1069.8	23.7
Suspended solids	138.5	108.6	8.4	1.2	99.1
TOC	121.1	121.1	24.3	12.1	90.0
Kjeldahl-N	17.0	15.3	7.2	4.6	72.9
TNT	178.2	175.7	14.7	3.7	97.9
RDX	145.2	148.9	30.1	19.5	86.6
Color	-	-	10.0	8.0	-

a. LAP = load, assemble, and pack areas of plant.

b. Adapted from Sullivan et al. 1979, citing Patterson et al. 1976.

c. TOC = total organic carbon; TNT = 2,4,6-trinitrotoluene; RDX = hexahydro-1,3,5-trinitro-1,3,5-triazine.

d. Diat. = diatomaceous (silicon containing) earth.

e. All values are averages with units of mg/L except pH and color. Color in Platinum-Cobalt Units (PCU).

TABLE 8.49. SUMMARY OF WASTEWATER QUALITY DATA AT IOWA ARMY AMMUNITION PLANT[a]

Parameter[b] (mg/L)	Precolumn Line 3A[c]	Carbon Column Line 3A[d]
TNT	175	0.58
RDX	61	0.55
Nitrite	1.8	2.4
Nitrate	24	14
pH	7.76	7.88
TOC	92	4.6

a. Adapted from Spanggord et al. 1978.

b. TNT = 2,4,6-trinitrotoluene; RDX = hexahydro-1,3,5-trinitro-1,3,5-triazine; TOC = total organic carbon.

c. Values are for influent.

d. Values are for effluent.

8.4.2 Air Quality

One report dealing with the impact on air quality from the manufacture of TNT and/or other munitions was a study done by Ghassemi et al. (1976). They looked at Joliet AAP and determined the amounts of released pollutants from the various stages of production (see Section 8.1.2.1). These values were compared to pollution standards for the state of Illinois (Table 8.7) and proposed Ammunition Procurement and Supply Agency (APSA) standards (Table 8.50). The atmospheric releases from JAAP at that time were in excess of (up to 15 times) the state standards and the APSA standards. The releases were also far above the ambient levels in the local atmosphere (Table 8.51). In both cases the major offending pollutants were nitrogen oxides, particulates, and acid mists. Sulfur dioxides were not as great a problem.

As part of a larger evaluation of AAP-associated air pollutants, Carpenter et al. (1978) compared the emissions released from Radford and Volunteer AAPs with the ambient air quality of their surrounding counties. The impact of the AAP depended on the industrial nature of the county (Table 8.52). In this case, VAAP does not contribute as large a

TABLE 8.50. EXISTING AIR POLLUTANT EMISSIONS FOR JOLIET ARMY AMMUNITION PLANT VS PROPOSED APSA[a] GUIDELINES[b]

Pollutants	APSA Guidelines (Stack Emissions)	Existing Emissions
Acid mist	50 mg/M^3	SAC[c] 205 mg/M^3 Sellite plants 2,500 mg/M^3
Ammonia	100 ppm	-
Hydrocabons	200 ppm	-
Hydrogen sulfide	100 ppm	-
Nitrogen oxides	200 pm	AOP[d] 2213 ppm Red water incinerators 1,730 ppm TNT, acid recovery 32,065 ppm TNT, fume recovery 9,500 ppm
Particulates	0.087 grain/cu ft (200 g/M^3)	South power plant 2.28 grain/cu ft
Particulates	0.197 grain/cu ft (450 g/M^3)	Red water incinerators 0.293 grain/cu ft

a. Ammunition Procurement and Supply Agency (U.S. Army).

b. Adapted from Ghassemi et al. 1976.

c. SAC = sulfuric acid concentrators.

d. AOP = ammonia oxidation plant.

TABLE 8.51. TYPICAL AMBIENT AIR MONITORING DATA[a] IN VICINITY OF JOLIET ARMY AMMUNITION PLANT[b]

Sampling Site (Designation)	NO_2 (ppm)	SO_2 (ppm)	Sulfation Rate (mg/100)	Dust Fall (tons/sq mile)	Suspended Particulates ($\mu g/M^3$)
Laboratory	-	0.006	0.008	-	52.5
Patrol road	0.001	0.001	0.012	30.9	58.5
Railroad office	0.001	0.001	0.008	21.9	44.8
Reservoir	0.001	0.001	0.007	17.0	99.1
Property disposal	0.001	0.001	0.008	19.4	72.7
Brown Circle	0.012	-	-	-	82.3
Des Plaines	0.001	0.001	0.009	32.0	31.3
Blodgett	0.001	0.001	0.007	15.4	-

a. Average of weekly samples for April 1973.

b. Adapted from Ghassemi et al. 1976.

percentage of air pollutants as does RAAP. However when the output of the VAAP is compared to an industrial power plant with similar nitrogen oxide emissions, a plant of 38 megawatt capacity is considered equivalent. This indicates that the emission output is significant and could have a considerable impact on the local environment. A similar analysis of the impact of an RDX/HMX/TNT plant on the associated county was also compiled for Holston AAP (Table 8.53). Once again the impact of HAAP was significant and equal to that of a 360-megawatt coal-fired power plant, even though the percentage of the total county emissions was small. Carpenter et al. did further analysis of the HAAP data and, using a computer model, calculated the concentrations of air pollutants at several boundary locations (Table 8.54). These ambient levels were based on emission data from RDX manufacture (see Table 8.11) and include dispersion factors. In comparison with EPA standards, only nitrogen oxides exceed acceptable levels, but they do so by a factor of 55 times. This further indicates the potential for significant impact of the emissions on air quality.

8.4.3 Soil and Groundwater Quality

Very little was reported in the literature regarding effects from exposure to TNT or other munitions on the quality of soil structure and groundwater parameters. The majority of data dealt with concentrations of the munition compounds in these media. Some aspects of this are discussed in the sections on persistence and transportation (see Sections 8.2.3.1 and 8.3.1.1), and the following discussion will primarily focus on levels of munitions found in soils and groundwater.

TABLE 8.52. COMPARISON OF RADFORD (RAAP) AND VOLUNTEER (VAAP) ARMY AMMUNITION PLANT EMISSIONS TO COUNTYWIDE EMISSIONS[a]

Sample Location	Particulates (ton/yr)	SO_2 (ton/yr)	CO (ton/yr)	NO_x (ton/yr)	HC[b] (ton/yr)
RAAP[c,d]	13,883	6,357	294	7,933	360
Montgomery Co.[c,e]	16,449	7,924	21,251	11,130	4,349
Percent[f]	85	80	1.4	72	8
VAAP[c]	36	74[h]	---	177	---
(RTI Estimate)[g]	37	528[h]	14	1,251	22
Hamilton Co.[c,e]	14,392	10,165	119,070	16,002	24,545
Percent[f]	0.2	0.7	0	1.1	0

a. Adapted from Carpenter et al. 1978.

b. HC = hydrocarbons

c. Data were retrieved from EPA's National Emissions Data System (NEDS) on June 7, 1977. NEDS is a computerized data bank which holds detailed emissions inventory data for each county in the country. It should be noted that differences in practices of reporting and updating emissions data may prevent comparisons on the same basis.

d. Emission rate is for 1972.

e. Emission rate as of June 2, 1971.

f. This value is the percent contribution of the army installation to countywide emissions.

g. Research Triangle Institute's emissions estimate at six 50 ton/day operations.

h. An additional 109 ton/year of H_2SO_4 mist are also indicated.

One report that provided data on groundwater quality was based on a study of the Alabama AAP, a facility that produced munitions between 1942 and 1945, and was maintained in standby condition until 1973 (Keirn et al. 1981). The groundwater sampled at AAAP did show some effects from the TNT production, including sizeable increases in levels of nitrate/nitrite-nitrogen, and smaller increases in specific conductance and sulfates (Table 8.55). Two sites had elevated levels of munitions and munition decomposition products (Table 8.55) that were traced to production facilities. The groundwater of Milan AAP was also evaluated for effects on the water quality (Envirodyne Engineers, Inc., 1980). Samples taken from various wells on the site indicated selected areas of groundwater contamination with detectable levels of TNT, RDX, and their degradation products (Table 8.56). Elevated levels were also found for nitrates, nitrites, sulfate, and phosphate (Table 8.56).

TABLE 8.53. COMPARISON OF HOLSTON ARMY AMMUNITION PLANT (HAAP) EMISSIONS TO COUNTYWIDE EMISSIONS[a]

Sample Location	Particulates (ton/yr)	SO_2 (ton/yr)	CO (ton/yr)	NO_x (ton/yr)	HC[b] (ton/yr)
HAAP[c]					
Area A[c,d]	1,108	2,209	217	1,726	1,782
Area B[c,e]	1,592	2,879	208	3,495	1,038
Total[c]	2,700	5,088	425	5,221	2,820
RTI estimate[f]	2,692	3,097	2,226	11,776	4,500[g]
Sullivan Co.[c]	31,024	42,289	71,009	28,965	23,350
Hawkins Co.[c]	19,116	62,541	15,139	38,594	4,469
Total[c]	50,140	104,830	86,148	67,559	27,819
Percent[h]	5.4	4.9	0.5	7.7	10.1

a. Adapted from Carpenter et al. 1978.

b. HC = hydrocarbons.

c. Data were retrieved from EPA's National Emissions Data System (NEDS) on June 7, 1977. NEDS is a computerized data bank which holds detailed emissions inventory data for each county in the country. It should be noted that differences in practices of reporting and updating emissions data may prevent comparisons on the same basis.

d. Located in Sullivan Co.

e. Located in Hawkins Co.

f. Research Triangle Institute's emissions estimate assumes full mobilization.

g. Includes 3,969 ton/yr of organic solvents.

h. This value is the percent contribution of the army installation to countywide emissions for the two-county area, with HAAP not at mobilization.

Keirn et al. (1981) also reported the levels of munitions found in the soil of the Alabama AAP (Table 8.57). The levels of various TNT-derived compounds were quite high in some localities. In particular, the former manufacturing areas and the landfill (at depth) contained high concentrations of TNT and many of its degradation products. When it is considered that the manufacture of TNT stopped at this site more than 30 years prior to sampling, the long-term potential of the munitions adversely affecting the soil quality is indicated. The evaluation of the Milan AAP included data on soil contamination by munitions and associated compounds (Envirodyne Engineers, Inc., 1980). Table 8.58 indicates the extent of the contamination and the degree with which it

TABLE 8.54. MAXIMUM "GROUND LEVEL" CONCENTRATIONS OF AIR POLLUTANTS NOTED AT BOUNDARY[a,b] OF HOLSTON ARMY AMMUNITION PLANT[c]

Compound	Area A ($\mu g/m^3$)	Area B ($\mu g/m^3$)	Total ($\mu g/m^3$)	Total (ppm)
Group I. EPA Criteria Pollutants				
Particulates	141.5	9.93	151.4	
Sulfur oxides	134.4	33.4	167.8	6.4×10^{-2}
Carbon monoxide	403.3		403.3	3.52×10^{-1}
Nitrogen dioxide	220.8	4,872	5,093	2.72
Nonmethane hydrocarbons	5.32	17.0	22.3	3.42×10^{-3}
Group II. Organics	(136.50)[d]	(1,984)[d]	2,120	1.94[e]
Acetic acid	1.71	1,807	1,809	5.76×10^{-1}
Acetic anhydride		87.7	87.7[f]	2.10×10^{-2}
Formic acid		13.4	13.4	7.14×10^{-3}
Isobutyl acetate		1.48×10^{-1}	1.48×10^{-1}	3.12×10^{-5}
n-Propyl acetate	40.1		40.1	9.63×10^{-3}
n-Propyl formate	2.20		2.20	6.13×10^{-4}
Methyl acetate	2.47	6.90	9.37	3.10×10^{-3}
Cyclohexanone		153	153	3.32×10^{-2}
Acetone		187	187	7.90×10^{-2}
Methyl ethyl ketone		4.22×10^{-1}	4.22×10^{-1}	1.67×10^{-4}
Methyl nitrate	1.10	119.1	120.2	3.32×10^{-2}
Nitromethane	2.09×10^{-1}	2	2	
Toluene		3.05	3.05	8.11×10^{-4}
Phenol	2.65×10^{-2}		2.65×10^{-2}	6.91×10^{-6}
Trace organics (butanol, propanol, methanol, methyl formate, formaldehyde)	88.64	6.11×10^{-1}[f]	89.25	1.36×10^{-1}[g]
Group III. Miscellaneous species				
Methane	64.0		64.0	9.86×10^{-2}
Hydrogen	13.8		13.8	1.69×10^{-1}
Carbon dioxide	47,760	15,050	62,810	35.0
Ammonia		38.8	38.8	5.60×10^{-2}
Nitric acid		550.3	550.3	2.14
Explosives (particulates)		1.65×10^{-1}[h]	1.65×10^{-1}[h]	

a. "ground level" = 1.5 m.

b. Assumes full mobilization

c. Adapted from Carpenter et al. 1978.

d. Values are totals.

e. ppm carbon.

f. The ambient concentrations ($\mu g/m^3$) for a particular compound should be consistent with the emission rate, varying somewhat with source height, velocity, and temperature. The value of the ratio between the emission rate (ton/year) and ambient concentration ranges from 2 to 7 for all the emitted compounds except acetic anhydride and trace organics. For acetic anhydride and trace organic emissions, this ratio has values of 0.5 and 0.02, respectively. It is believed that the calculated ambient concentration of these compounds is higher than it actually would be.

g. Calculated using the molecular weight of methane.

h. Consists of 6.6×10^{-2} $\mu g/m^3$ hexahydro-1,3,5-trinitro-1,3,5-triazine and 9.86×10^{-2} $\mu g/m^3$ 2,4,6-trinitrotoluene.

TABLE 8.55. SUMMARY OF GROUNDWATER QUALITY DATA AT ALABAMA ARMY AMMUNITION PLANT (AAAP)[a]

Sample Location[h]	Sp. Cond.[b] (umho/cm)	pH	Nitrate/Nitrite (ppm as N)	Sulfate (ppm)	2,4-DNT[c] (ppb)	2,6-DNT[d] (ppb)	TNT[e] (ppb)	TNB[f] (ppb)	DNP[g] (ppb)
Background values	74-180	5.9-6.2	<0.1-3.23	<7-23					
LBA, P-1	180	6.1	0.189	23	ND[i]	ND	ND	ND	ND
LBA, P-2	74	5.9	2.49	<7	ND	ND	ND	ND	ND
LBA, P-3	768	6.7	<0.10	41	ND	ND	ND	ND	ND
IND, P-4	33	6.1	0.408	<7	ND	ND	ND	ND	ND
IND, P-5	282	6.3	0.346	40	ND	ND	ND	ND	ND
IND, P-6	150	6.4	0.117	19	ND	ND	ND	ND	ND
IND, P-7	76	6.0	<0.100	8	ND	ND	ND	ND	ND
IND, P-8	384	6.7	<0.100	24	ND	ND	ND	ND	ND
IND, P-9	492	8.2	<0.100	25	ND	ND	ND	ND	ND
IND, P-10	354	5.4	25	29	4,340	860	10,270	4,380	1,100
IND, P-11	382	6.8	1.53	45	21	38.7	74.4	<5.3	ND
IND, P-12	232	6.2	0.891	<7	ND	ND	ND	ND	ND
IND, P-13	552	6.5	10.5	49	ND	ND	ND	ND	ND
IND, P-14	186	6.2	0.478	27	ND	ND	ND	ND	ND
IND, P-15	122	6.0	0.330	16	ND	ND	ND	ND	ND
IND, P-16	40	5.8	0.421	<7	ND	ND	ND	ND	ND
IND, P-17	389	6.3	<0.100	45	ND	ND	ND	ND	ND
IND, P-18	195	6.1	0.402	<7	ND	ND	ND	ND	ND
IND, P-19	235	6.2	1.10	61	ND	ND	ND	ND	ND
IND, P-20	50	5.6	0.238	<7	ND	ND	ND	ND	ND
GSA, P-21	77	6.0	0.116	<7	ND	ND	ND	ND	ND
GSA, P-22	120	6.2	<0.100	8	ND	ND	ND	ND	ND
GSA, P-23	93	6.0	0.116	<7	ND	ND	ND	ND	ND
IND, P-25	58	5.9	0.232	<7	ND	ND	ND	ND	ND
IND, P-26	133	6.0	0.284	<7	ND	ND	ND	ND	ND

a. Adapted from Keirn et al. 1980.

b. Sp. Cond. = specific conductance.

c. 2,4-DNT = 2,4-dinitrotoluene.

d. 2,6-DNT = 2,6-dinitrotoluene.

e. TNT = 2,4,6-trinitrotoluene.

f. TNB = 1,3,5-trinitrobenzene.

g. DNP = 2,4-dinitrophenol.

h. Areas of AAAP in which wells were located, LBA = Leaseback area; IND = industrial area (explosives manufacture, flashing grounds, Manhattan project area, sanitary landfill); GSA = General Services Administration area.

i. ND = not determined.

is associated with munition disposal areas. Levels were found for RDX up to 83,000 ng/g and for TNT up to 25,000 ng/g. The presence of large levels of RDX and TNT indicate that substantial amounts of the primary munitions have not undergone degradation. A similar situation was found by Jerger et al. (1976) at Iowa AAP in an abandoned waste lagoon site that had been filled in by natural processes and by the addition of coal-generated wastes. After 20 years of disuse, levels of TNT and by-products up to 3,030 mg/kg were found at this site. The physical nature of the site had also changed, with the ground being barren and a deep reddish color.

TABLE 8.56. SUMMARY OF GROUNDWATER QUALITY DATA AT MILAN ARMY AMMUNITION PLANT[a]

Parameter[b] (in µg/L)	Total Samples	Number of Positive Samples	Range	Average[c]	Detection Limits
Nitrate	44	44	<100-26,000	2,586	<100
Nitrite	44	44	<10-100	21	<10
Phosphate	44	44	<200-1,500	323	<200
Sulfate	44	44	<4,600-140,000	14,672	<4,600
2,4-DNT	39	5	<10-70	22	<10
2,6-DNT	39	1	5		
2,4,6-TNT	39	4	<10-3000	823	<10
1,3,5-TNB	29	1	1,100		<200
RDX	39	4	<20-780	270	<20

a. Adapted from Envirodyne Engineers, Inc. 1980.

b. 2,4-DNT = 2,4-dinitrotoluene; 2,6-DNT = 2,6-dinitrotoluene; 2,4,6-TNT = 2,4,6-trinitrotoluene; 1,3,5-TNB = 1,3,5-trinitrobenzene; RDX = hexahydro-1,3,5-trinitro-1,3,5-triazine.

c. Average is calculated with < values assumed to be equal to the value itself.

8.5 TOXIC EFFECTS OF TNT, RDX, HMX, AND ASSOCIATED COMPOUNDS

8.5.1 Toxic Effects of TNT and Metabolites on Organisms

8.5.1.1 Bacteria

Several references were found that evaluated the effects of alpha-TNT on bacteria. Often this aspect was ancillary to study of the microbial degradation possibilities. One such study was reported by Enzinger (1971). He used cultures of Zoogloea ramigera 115, a bacterial strain found in large numbers in sewage treatment facilities which possesses the ability to flocculate and adsorb metallic ions. The cultures were acclimated to and eventually grown in 100 ppm TNT broth for 120 hours. The bacteria were subject to TNT shock when introduced without acclimation to 100 ppm TNT solutions, and demonstrated morphological changes when exposed to concentrations greater than 30 ppm. The morphological changes consisted primarily of a shift from small rods (1.5 to 2.0 microns in length) to long filamentous rods (10 to 20 microns in length). These changes were found to be reversible. In a comparison of the abilities of Pseudomonas species and Escherichia coli to decompose TNT, Amerkhanova and Naumova (1975) state that there is no general correlation between decomposing ability and resistance to TNT toxicity; P. fluorescens was found to be highly resistant while Pseudomonas sp. I was rather sensitive. A study of bacteria in aquatic sediments downstream from Iowa AAP indicated that growth was not affected by TNT concentrations of 10 mg/L as long as other nutrients were also present

TABLE 8.57. SUMMARY OF MUNITION LEVELS IN SOILS AT ALABAMA ARMY AMMUNITION PLANT (AAAP)a

Sample Location (AAAP area)	Sample Depth (in cm)	Number of Samples	2,4-DNTb (ppb)	2,6-DNTc (ppb)	TNTd (ppb)	TNBe (ppb)	DNBf (ppb)
Magazine	10	28	160	NDh	ND	1,965	ND
Old burning grd	10	5	488	115	177	<369[i]	<170
Old burning grd	15	2	<112	<102	<37	<369	<170
Old burning grd	50	1	386	<102	<37	<369	<170
Old burning grd	100	1	766	471	90	<369	<170
Propellant shipping	10	1	370	ND	ND	ND	ND
Rifle powder finishing	10	10	1,757	ND	ND	ND	ND
Leaseback area	10	5	825	ND	ND	ND	ND
Leaseback area	10	1	825	ND	ND	615	ND
Burn./Sanitary landfill	10	3	<112	<102	<37	<368	<170
Burn./Sanitary landfill	100	1	<112	<102	<37	<368	<170
Burn./Sanitary landfill	230	1	<112	<102	1,170	1,530	<170
South. TNT manufacturing	10	11	152	166	273,066	459	<170
South. TNT manufacturing	100	1	793	4,840	120	<368	<170
North. TNT manufacturing	10	7	1,105	134	101,433	713	191
Flashing ground	10	7	572	<102	396	875	<170
Flashing ground	25	1	<112	757	<37	684	<170
Flashing ground	90	1	<112	<102	79	<368	<170

a. Adapted from Keirn et al. 1981.

b. 2,4-DNT = 2,4-dinitrotoluene.

c. 2,6-DNT = 2,6-dinitrotoluene.

d. TNT = 2,4,6-trinitrotoluene.

e. TNB = 1,3,5-trinitrobenzene.

f. DNB = 2,4-dinitrobenzene.

g. In samples of 2 or more, values are averages.

h. ND = not determined.

i. < Values represent the limit of detection for that munition; in averages, < numbers were assumed to be equal to the detection limit.

Table 8.58. SUMMARY OF MUNITION LEVELS IN SOILS AT MILAN ARMY AMMUNITION PLANT (MAAP)[a]

Sample Location (MAAP area[f])	2,4-DNT[b] (mg/g)	2,6-DNT[c] (mg/g)	TNT[d] (mg/g)	RDX[e] (mg/g)	Nitrate (µg/g)	Nitrite (µg/g)	Phosphate (µg/g)	Sulfate (µg/g)
Background-1A	-[g]	<0.2[h]	<2	<60	1.7	0.50	<0.5	30
Background-1B	-	<0.2	<8	-	1.5	0.10	<0.5	35
Background-1C	-	<0.2	5	<60	1.6	0.05	<0.5	20
Background-1D	-	<0.5	11	-	3.0	0.03	<0.5	18
Background-1E	-	<0.2	27	-	4.5	<0.03	<0.5	20
Background-2A	-	<0.2	<2	120	1.9	1.2	<0.5	30
Background-2B	-	1.1	3.7	160	1.7	0.20	<0.5	30
Background-2C	-	<0.2	2	<60	1.4	0.38	<0.5	30
Background-2D	-	<0.5	<8	-	1.4	0.18	<0.5	18
Background-2E	-	0.1	1.6	51	0.5	0.08	<0.5	18
BG, downwind-3A	-	-	67	770	0.90	0.05	0.6	25
BG, downwind-3B	-	1.4	6	200	1.0	0.05	0.8	30
BG, downwind-3C	-	<0.2	46	850	0.90	0.05	0.8	58
BG, downwind-3D	-	-	2	110	1.0	0.03	<0.5	20
BG, downwind-3E	-	<0.2	11	820	0.80	0.03	0.8	30
Old burning area-4A	-	-	65	<120	0.40	<0.03	0.5	25
Old burning area-4B	-	-	2	90	0.30	<0.03	<0.5	32
Old burning area-4C	-	29	140	83,000	2.7	<0.03	<0.5	30
Old burning area-4D	-	-	-	<2,200	0.70	<0.03	<0.5	25
Old burning area-4E	-	<0.7	230	6,000	0.70	<0.03	0.58	<12
Demolition area-5A	3	<0.5	23	-	3.5	<0.03	4	35
Demolition area-5B	34	2.1	78	310	2.2	<0.02	<0.5	38
Demolition area-5C	17	4	590	740	3.7	0.03	<0.5	25
Burning area-6A	<30	<5	110	<2,200	9.0	0.03	<0.5	55
Burning area-6B	580	87	9,500	13,000	9.1	0.25	<0.5	50
Burning area-6C	10	2	4,000	-	4.0	0.03	<0.5	32
Burning area-6D	600	320	4,400	<220	5.0	0.03	0.8	42
Pine forest, BC-7A	-	0.2	<2	200	0.78	0.22	3.8	25
Pine forest, BC-7B	-	<0.2	3	<60	3.9	<0.03	6.1	25
Pine forest, BC-7C	-	0.5	2	74	1.3	0.05	7.6	25
Pine forest, BC-7D	-	0.6	2	<60	4.3	0.15	4.4	20
Pine forest, BC-7E	-	0.2	<2	80	1.5	0.7	3.6	30
Old landfill marsh-8A	-	0.2	11	1,600	0.18	0.22	0.6	18
Old landfill marsh-8B	-	<2	<20	<600	0.45	<0.03	0.8	30
Old landfill marsh-8C	-	0.03	3.5	640	0.18	0.12	<0.5	42
Old landfill marsh-8D	-	2	40	6,100	0.50	<0.03	0.7	44
Old landfill marsh-8E	-	<2	20	46,000	0.50	<0.03	0.5	53
Lagoon spoil bank-9A	270	120	25,000	<220	12	0.22	<0.5	62
Lagoon spoil bank-9B	34	8	12,000	72,000	22	0.32	<0.5	75
Old nitrate spill-10A	-	-	30	<600	0.42	<0.03	<0.5	25
Old nitrate spill-10B	<0.8	0.9	16	110	0.60	<0.03	0.5	18
Old nitrate spill-10C	<0.8	-	29	70	0.28	0.03	<0.5	18
Old nitrate spill-10D	-	0.3	10	70	0.42	<0.03	<0.5	12
Old nitrate spill-10E	-	<0.5	10	-	0.30	0.03	<0.5	12
Old fertilizer spill-11A	-	0.7	7	5,300	0.50	<0.03	<0.5	30
Old fertilizer spill-11B	-	-	-	-	0.90	0.25	<0.5	18
Old fertilizer spill-11C	-	<2	20	5,400	0.82	0.03	<0.5	12
Old fertilizer spill-11D	-	-	62	32,000	0.60	0.30	<0.5	20
Old fertilizer spill-11E	-	-	24	620	0.30	0.05	<0.5	20

a. Adapted from Envirodyne Engineers, Inc. 1980.

b. 2,4-DNT = 2,4-dinitrotoluene.

c. 2,6-DNT = 2,6-dinitrotoluene.

d. TNT = 2,4,6-trinitrotoluene.

e. RDX = hexahydro-1,3,5-trinitro-1,3,5-triazine.

f. BG, Downwind = background area, downwind from burning facility; Pine Forest, BC = pine forest samples below bird colony.

g. - represents the absence of that compound.

h. < Values represent the presence of the compound below the limit of detection for that parameter.

(Weitzel et al. 1975). In an evaluation of a bacterial bioluminescence assay, Bulich et al. (1981) reported that TNT had a 5-minute EC_{50} of 20 mg/L for the bioluminescent bacterial strain Photobacterium phosphoreum. Bringmann and Kuehn (1977) tested Pseudomonas putida for a threshold effect using an inhibitory action as an indicator. They found effects at levels of 9 mg/L for 2,3-DNT, 26 mg/L for 2,6-DNT, 57 mg/L for 2,4-DNT, and >100 mg/L for 2,4,6-TNT.

One of the few studies designed primarily to evaluate the effects of TNT on bacterial populations was reported by Jerger et al. (1976). They evaluated the microbiology of stream sediments associated with two AAPs (Joliet and Iowa) using both field and laboratory studies. In the field, they evaluated the effects of TNT wastewater discharge by assessing changes in the parameters of bacterial enumeration, microbial inhibition, bentbic dissolved oxygen uptake, dehydrogenase activity, and ATP activity. Following these evaluations, they studied TNT toxicity on anaerobic systems in the laboratory. The field studies showed no differences between TNT exposed stations and controls even when some TNT sediment concentrations reached 44,200 mg/kg. The laboratory anaerobic degradation studies indicated that TNT concentrations of 100 ppm inhibited methane production initially, but after acclimation a concentration of 200 ppm was required to produce toxic effects.

Klausmeier et al. (1974) reported a study of the toxic effects of TNT on soil organisms, including bacteria and fungi. They found an inhibition of growth in pure culture and soil plate-count studies. The inhibition generally occurred at levels of 50 mg/L and above for bacteria and 20 mg/L and above for fungi. At lower levels, some degradation of TNT was indicated. Species tested included the gram-positive bacteria, Bacilius and the fungi genera Penicillium, Phycomyces, Trichoderma, Aspergillus, Chaetomium, Myrothecium, Fusarium, Mimnoniella, and Rhizopus. In an evaluation of the effect of TNT on the decomposing ability of the soil organisms, a 1 percent concentration was found to inhibit the decomposition of filter paper and polyvinyl plastic.

8.5.1.2 Aquatic Algae, Phytoplankton, and Vascular Plants

The effects of TNT on aquatic algae were discussed sporadically in the literature. In a review paper, Jaffe et al. (1973) mentioned a 1952 study of TNT toxicity to blue-green algae by Fitzgerald, who found that an 8-ppm solution killed 100 percent of Microcystis aeruginosa cells in suspensions containing 1-2 x 10 cells/mL salt solution. Jaffe et al. pointed out that some protocol details were not provided for this study, including time to death, means of determining death, and whether an assay to determine the actual levels of TNT in the test solution was performed.

Field surveys of some of the AAPs (Huff et al. 1975a, 1975b) generally were not informative on algae/TNT interaction because of difficulty in determining which munition or waste was responsible for any effects detected and the difficulty in sampling a constantly changing ecosystem component like the phytoplankton community. Weitzel et al.

(1975), in an aquatic field survey of Iowa AAP, did find some shifts in periphyton species diversity, chorophyll a production, and organic mass that corresponded to distance from the plant and to TNT levels in the streams. However, the evidence was not conclusive in correlating the changes with TNT levels alone since other chemical and biological factors could have produced similar changes in the measured parameters. In another study of Iowa AAP, Sanocki et al. (1976) found some correlation between TNT levels at stream stations and the periphyton community on artificial substrates. Generally, higher TNT levels resulted in lower species diversity. These effects were short-term in nature, with recovery demonstrated at the sites most downstream.

A similar analysis of the stream periphyton communities in the area of the Joliet AAP by Stilwell et al. (1976) indicated toxic effects of TNT containing wastewater (pink water). Their survey indicated no effects on the periphyton community during the spring samples, primarily due to the continuous flow of the system during this period. However during the fall, the flow rate decreased significantly and effects were noted. Below the wastewater discharge, the number and diversity of species were reduced on both artificial and natural substrates. The species composition changed from a nonpolluted, low tolerance community above the discharge point to a pollutant-tolerant community below the discharge site. Biomass, chlorophyll a, and autotrophic index parameters also supported this pattern. The effects were due in part to the input of TNT wastewater (containing isomers of dinitrotoluene as well as TNT), but an industrial waste effluent discharge site also contributed to the negative effects on the periphyton community. Thus, a clear assessment of the effects of TNT wastes could not be drawn from this study despite the demonstrated effects on the periphyton community.

Putnam et al. (1981) evaluated the toxic effects of TNT wastes from the Volunteer AAP on the periphyton population of Waconda Bay. They used artificial substrates (glass microscope slides) and samples from natural substrates and determined effects on biomass and diatom species composition (community structure). These parameters were then related to TNT waste concentrations in the water. During the winter, the zone of influence extended down the bay, with population and biomass inversely related to the concentration of VAAP wastes. The diatom population ranged from $27/mm^2$ at the site closest to VAAP to $37,267/mm^2$ at one of the reference sites. The pattern of chorophyll a content at the sites indicated toxic suppression over the first kilometer downstream. This was followed by a stimulatory effect over the next kilometer and finally an intermediate leveling off effect at the last two stations. This suggests that a dilution effect or degradation process detoxifies the TNT waste, and a nutrient stimulation effect occurs. In relation to observed and calculated (based on effluent concentrations and dilution factors) TNT waste levels, the periphyton data indicate that no effects occurred at levels lower than 25 µg/L, definite effects occurred at 500 to 600 µg/L, and minimal effects were associated with levels of 50 to 100 µg/L.

More conclusive evidence of the direct link between TNT and toxic effects to algae species was supplied by Smock et al. (1976). They analyzed the growth rates of cultures of the green alga Selenastrum capricornutum and the blue-green alga Microcystis aeruginosa over a 17-day period using dose concentrations ranging from 1 to 50 mg/L TNT. Concentrations of 5 mg/L inhibited growth of S. capricornutum cultures while a 15 mg/L concentration limited growth in M. aeruginosa cultures. They also tested pink water complexes and found that levels above 9 mg/L inhibited S. capricornutum growth and a 50 mg/L level was lethal to M. aeruginosa. Lower levels stimulated growth of the M. aeruginosa cultures. Additional responses of the M. aeruginosa populations were production of gas vacuoles and a gelatinous sheath which resulted in a floating algal mat formation. The occurrence of these effects was in proportion to the TNT concentrations. The authors speculated that this might be a defense response to the toxic effects of the TNT solutions since the sheaths added some surface shielding, and the mat formation helped isolate the algae.

Won et al. (1976) used the green algal species, Selenastrum capricornutum, to test TNT and some by-products for toxic effects. The by-products included 2,6-dinitro-4-hydroxyaminotoluene, 2,6-dinitro-4-aminotoluene, 2-nitro-4,6-diaminotoluene, 2,4-dinitro-6-hydroxyaminotoluene, 2,4-dinitro-6-aminotoluene, 2,2',4,4'-tetranitro-6,6-azoxytoluene, and 2,2',6,6'-tetranitro-4,4'-azoxytoluene. Cultures grown for 7 days in the presence of the test compounds were evaluated for growth and morphological effects. Only TNT produced any effects, with a concentration as low as 2.5 mg/L suppressing growth, causing chlorosis, and producing a population dominated by ballooned, granulated cells.

Another study examining the effects of TNT and some of its by-products on algal species was reported by Bringmann and Kuehn (1978). They determined the threshold value for inhibition of cell propagation in blue-green algae, Microcystis aeruginosa, and green algae, Scenedesmus quadricauda, when exposed to test solutions of 2,4,6-TNT, 2,4-DNT, 1,3-dinitrobenzene, 2,3-DNT, and 2,6-DNT. When exposed to the above substances, M. aeruginosa showed inhibition at 0.32 mg/L, 0.13 mg/L, 0.17 mg/L, 0.22 mg/L, and 0.50 mg/L, respectively. For S. quadricauda the inhibition values were 1.6 mg/L, 2.7 mg/L, 0.70 mg/L, 0.83 mg/L, and 12 mg/L, respectively. The inhibition values for both species show that some of the by-products of TNT can be more toxic than the parent compound. Thus, in the environment, equal concern must be placed on the effects of the breakdown products and those of the major compound.

Evaluation of the toxic effects to aquatic vascular plants of TNT and its breakdown products was restricted to a study by Schott and Worthley (1974) using the common duckweed Lemna perpusilla. They evaluated the effects of several compounds on the growth of duckweed in test flasks. A reduction in growth rate of 10 percent after 11 days was considered to be significant. At a pH of 6.3, they found a significant growth reduction at concentrations of 1 ppm for 2,4,6-TNT, 0.5 ppm for

2,4-DNT, and 50 ppm for 4-amino-2-nitrotoluene (Table 8.59). At a pH of 8.5, the effective level changed to 10 ppm for 4-amino-2-nitrotoluene. Above these dose levels the response was death.

8.5.1.3 Aquatic Invertebrates

Two general types of information on TNT effects on aquatic invertebrate species were found in the literature: field data on community level effects and laboratory studies on the effects to individual species.

In a series of studies, Huff et al. (1975a,b,c,d) sampled the aquatic systems in the vicinity of four AAPs for effects on zooplankton and macroinvertebrate populations. They looked at numbers of species and individuals, species diversity (pollution tolerant or intolerant), and community diversity (Shannon-Weaver indexes). Zooplankton and macroinvertebrates (primarily benthic) were sampled in fall collections. The zooplankton samples were inconclusive at three riverine sites (Radford, Milan, and Holston) and showed no effects at one site. This site, Volunteer AAP, discharged into a lake-type ecosystem which allowed more extensive sampling, avoiding a problem associated with the other AAP sites. The lake environment also would allow the zooplankton to migrate and avoid high concentrations of TNT but still remain in the same general habitat. The macroinvertebrate populations were easier to characterize, and Huff et al. found indications at three sites (Radford, Holston, and Volunteer) of deleterious effects. At Radford, the number of taxa, specimens, and diversity all decreased below the waste outfalls when compared to an upstream control, and recovery of the parameters was evident in the two most downstream stations. The Holston AAP showed decreases in diversity at downstream stations, but industrial discharges complicated the analysis of TNT effects. Similar effects were noted at

TABLE 8.59. SUMMARY OF Lemna perpussila COLONY GROWTH WHEN EXPOSED TO VARIOUS NITROTOLUENES[a]

Compounds	pH	Plant Responses[b] Concentrations (ppm)								
		100	50	10	5	1	0.5	0.1	0.01	0.001
4-Amino-2-nitrotoluene	6.3	D	X	0	-	0	-	-	-	-
	8.5	X	X	X	-	0	-	-	-	-
2,4-Dinitrotoluene	6.3	D	-	D	-	X	X	0	0	-
	8.5	D	D	D	D	X	-	0	-	-
2,4,6-Trinitrotoluene	6.3	-	D	D	D	X	0	0	0	-
	8.5	-	D	D	D	X	-	0	-	-

a. Adapted from Schott and Worthley 1974.

b. D = death; X = decrease in growth rate; 0 = no effect; - = not tested.

Volunteer AAP, with the benthic community of the bay habitat showing reduced diversity near the outfall and recovery at distant stations. At Milan AAP, no effects were found due to poor habitat and therefore a lack of a suitable population for evaluation.

The macroinvertebrate populations at Volunteer AAP were also evaluated by Putnam et al. (1981). They looked at species numbers and diversity on artificial (Hester-Dendy plates) and natural substrates. The two most common organisms were Chironomids and Oligochaetes, with Chironomids dominating on artificial plates and the Oligochaetes in sediment samples. The highest densities and numbers of species were found in the reference samples, indicating some suppression at all of the downstream sites. The first five sites (up to 1 km downstream from VAAP) averaged 3.3 species, sites over the next kilometer averaged 9.5, and the reference sites averaged 13.5 species. The same general pattern of response to TNT is shown for the macroinvertebrates as for the periphyton data (Section 8.5.1.2). The no-effect level for the macroinvertebrates was 25 µg/L; minimal effects occurred at 50 to 100 µg/L, and more pronounced effects occurred at 500 µg/L and above.

Three additional field studies were conducted at two other AAPs, Iowa and Joliet. These studies looked at similar indices of population level effects but only on the macroinvertebrates. Weitzel et al. (1975) studied the Iowa AAP site in late summer and found inconclusive results for adverse effects on species numbers and diversity. A study of the same site by Sanocki et al. (1976) in the spring and fall also found no pattern of change in numbers of individuals, but the diversity of species was associated with TNT levels, especially for levels in sediments. The third study at Joliet AAP was also conducted in the spring and fall (Stilwell et al. 1976). The authors found evidence of toxic effects of TNT for both numbers and diversity of species. These effects were primarily associated with levels of pink water constituents in excess of 110 ppb. An increase of effects with higher levels of TNT in sediments was also found. The pattern of effects and their relation to the TNT waste discharges of the AAPs indicate toxicity of TNT to aquatic invertebrates. However uncertainity was introduced into the results of the field studies by confounding factors, such as the presence of other industrial wastes, degradation of habitats by dredging or channelizing, and difficulty in sampling. Thus, laboratory studies were needed to tighten the association indicated by the field studies.

The available laboratory studies of the toxicity of TNT and its by-products were limited to acute tests designed to generate LC_{50} values. Table 8.60 gives the results from several acute studies on invertebrate species. Daphnia magna, a common laboratory invertebrate test species, was found to have TNT LC_{50} values of 6.6 and 11.9 mg/L in static tests (Liu et al. 1976; Pearson et al. 1979). Won et al. (1976) used two saltwater invertebrates and determined approximate TNT LC_{50} values between 5 and 10 mg/L. Similar data were also found for some of the by-products (degradation or manufacturing impurities) of TNT. Liu et al. (1976) recorded an LC_{50} value of 35.0 mg/L for D. magna when exposed to 2,4-DNT under the same test conditions used to study TNT. Pearson et al. (1979) in static 48-hour tests using D. magna determined LC_{50} values

TABLE 8.60. ACUTE TOXICITY TESTS OF TNT IN AQUATIC INVERTEBRATE SPECIES

Species Name	Assay Type[a]	pH/Temperature[b]	LC_{50}[c]	Reference
Daphnia magna (waterflea)	48 hr, static	7.0/20°C	6.6	Liu et al. 1976
Daphnia magna (waterflea)	48 hr, static	?/?	11.9	Pearson et al. 1979
Tigriopus californicus (tidepool copepod)	72 hr, static	?/20°C	~5[d]	Won et al. 1976
Crassostrea gigas (oyster larvae)	96 hr, static	?/20°C	>5,<10[d]	Won et al. 1976

a. Static assay indicates no renewal of water or toxic compound.

b. pH value given first.

c. LC_{50} value is given in mg/L as determined by Litchfield and Wilcoxon method.

d. LC_{50} values are approximate and were not determined by Litchfield and Wilcoxon method.

(mg/L) of 35.0 for 2,4-DNT, 4.7 for 2,3-DNT, and 0.69 for 2,3,6-TNT. Values for tests of other by-products are given in Table 8.61 (Pearson et al. 1979). Bringmann and Kuehn (1982) determined an EC_{50} (effective concentration) level of <10 mg/L for D. magna exposed to 2,3-DNT. Those studies indicate that TNT and associated compounds do have a toxic effect on invertebrates but not at extremely low levels. Only 2,3,6-TNT, an isomer of alpha-TNT, produced a low LC_{50} value. However, these studies give no indication of the chronic effects and little indication of effects on larger, nonplanktonic, invertebrate species.

8.5.1.4 Fish

The literature on toxicity of TNT to fish species contained data from field studies and acute laboratory tests. The field studies consisted of surveys conducted at several AAP sites and were designed to evaluate species abundance and diversity. Three studies, at Milan, Holston, and Radford AAPs, revealed no conclusive proof of toxic effects of TNT on fish because of the same interfering factors as found in the invertebrate studies (Huff et al. 1975a,c,d). At Milan the dredged and channelized streams eliminated the majority of the fish habitat and at Holston the presence of HMX and RDX wastes as well as industrial wastes complicated the association of TNT levels and effects on fish. The study at Radford AAP did indicate some effects of TNT on species abundance and diversity, but because of differences in habitat types between sampling stations and poor sampling efficiency the evidence is not conclusive. This study also indicated recovery of the fish population at the most downstream site. At the Volunteer AAP, Huff et al. (1975b) found that after a period of 3.5 days of waste discharge, the fish abundance and diversity decreased in comparison to earlier samples. The most indicative evidence from a field study of toxic effects on fish was reported by Weitzel et al. (1975) for the Iowa AAP. They found an

TABLE 8.61. ACUTE TOXICITY VALUES OF TNT CONDENSATE WATER COMPONENTS[a]

Compound	Fathead Minnows[b] 96-h LC_{50} (mg/L)	*Daphnia magna* 48-h EC_{50} (mg/L)
Toluene	12.6	19.6
2-Nitrotoluene	38.0	>77.1[c]
4-Nitrotoluene	49.9	12.1
2,3-Dinitrotoluene	1.9	4.7
2,4-Dinitrotoluene	32.5	35.0
2,5-Dinitrotoluene	1.3	3.4
2,6-Dinitrotoluene	19.8	21.7
3,4-Dinitrotoluene	1.5	3.1
3,5-Dinitrotoluene	22.0	45.1
2,3,6-Trinitrotoluene	0.12	0.69
2-Amino-4-nitrotoluene	71.3	22.5
2-Amino-6-nitrotoluene	49.9	13.2
3-Amino-4-nitrotoluene	25.5	5.8
4-Amino-2-nitrotoluene	26.1	14.2
2-Amino-4,6-dinitrotoluene	14.8	4.5
2-Amino-3,6-dinitrotoluene	0.78	2.2
3-Amino-2,4-dinitrotoluene	12.2	8.1
3-Amino-2,6-dinitrotoluene	11.3	4.7
4-Amino-2,6-dinitrotoluene	6.9	5.2
4-Amino-3,5-dinitrotoluene	>13.1[c]	>13.1[c]
5-Amino-2,4-dinitrotoluene	2.4	3.1
1,3,5-Trinitrobenzene	1.03	2.7
1,3-Dinitrobenzene	7.4	53.0
1,5-Dimethyl-2,4-dinitrobenzene	7.9	24.3
3,5-Dinitroaniline	21.4	14.7
3-Methyl-2-nitrophenol	46.1	18.8
5-Methyl-2-nitrophenol	47.0	21.3
2,4-Dinitro-5-methylphenol	3.2	3.5
4-Nitrobenzonitrile	24.4	49.4
3-Nitrobenzonitrile	60.2	48.1

a. Adapted from Pearson et al. 1979.

b. *Pimephales promelas*.

c. Not toxic to 50 percent or greater of the organisms at its solubility limit.

increase in species diversity with distance from the waste outfalls. Also samples taken in a tributary creek that ran from the waste outfall to the main river, and therefore contained the highest levels of TNT, contained only six individuals of one fish species.

More evidence of the toxic effects of TNT was provided from laboratory tests. The earlier studies of TNT toxicity to fish usually did not

identify the species or test conditions used (Jaffe et al. 1973), and the data they generated did not add any more significant information than the more recently performed studies discussed below.

Pederson (1970) tested TNT for acute toxic effects on the bluegill Lepomis macrochirus at different water temperature and hardness levels. As shown by results in Table 8.62, temperature affected the LC_{50} values. The tests of water hardness produced identical LC_{50} values at 60 or 180 ppm (as $CaCO_3$) as long as the temperatures were equal. A 96-hour mean tolerance limit of 2.6 mg/L was determined by Nay et al. (1974) for the bluegill in a static bioassay. The LC_{50} values for the bluegill ranged from 2.3 to 2.8 mg/L over a temperature range of 10 to 25°C.

TABLE 8.62. ACUTE TOXICITY TESTS OF TNT IN FISH SPECIES

Species Name	Assay Type[a]	pH/Temperature[b]	LC_{50}[c]	Reference
Lepomis macrochirus (bluegill)	96 hr, static	6.5/25°C	2.8	Pederson 1970
Lepomis macrochirus (bluegill)	96 hr, static	6.5/10°C	2.3	Pederson 1970
Lepomis macrochirus (bluegill)	96 hr, static	7/7	2.6[d]	Nay et al. 1974
Pimephales promelas (fathead minnow)	24 hr, static	5.0/20°C	4.2	Liu et al. 1976
Pimephales promelas (fathead minnow)	24 hr, static	7.0/20°C	>3.2	Liu et al. 1976
Pimephales promelas (fathead minnow)	24 hr, static	9.4/20°C	3.0	Liu et al. 1976
Pimephales promelas (fathead minnow)	96 hr, static	5.0/20°C	1.2	Liu et al. 1976
Pimephales promelas (fathead minnow)	96 hr, static	7.0/20°C	2.0	Liu et al. 1976
Pimephales promelas (fathead minnow)	96 hr, static	9.4/20°C	2.4	Liu et al. 1976
Pimephales promelas (fathead minnow)	96 hr, dynamic	7/24°C	2.58	Smock et al. 1976
Pimephales promelas (fathead minnow)	96 hr, static	7/7	2.4	Pearson et al. 1979
Xiphorus helleri (green swordtail)	116 hr, semistatic	7/20-24°C	<5	Klausmeier et al. 1982

a. Static assay indicates no renewal of water or toxic compound, dynamic assay indicates continual flow-through of water and toxic compound, and semistatic assay indicates some renewal of water or test compound.

b. pH value given first.

c. LC_{50} value is given in mg/L as determined by Litchfield and Wilcoxon method.

d. Value is a TLm; mean tolerance limit.

Several studies utilized the fathead minnow Pimephales promelas as the test species. Liu et al. (1976) generated a range of LC_{50} values for various pH levels at 20°C, using both a 24- and 96-hour static acute tests. In the 96-hour tests, LC_{50} values increased with a higher pH, while in the 24-hour tests the values were lower at a higher pH (Table 8.62). Smock et al. (1976) also used the fathead minnow as the test species in a 96-hour dynamic acute test of TNT toxicity at 24°C. They generated an LC_{50} value of 2.58 mg/L (Table 8.62). They also looked at behavioral responses to exposure and determined an EC_{50} value of 0.46 mg/L for a moribund response criteria. The exposed fish exhibited three main behavioral responses to TNT. The initial response indicated shock as the fish began to gasp at the surface and move lethargically. A loss of motor control indicated the second phase with typical behavior including a jerky, surface-swimming motion and rapid gill movement. The third phase consisted of a slow yawing, pitching movement indicating some loss of equilibrium. The behavioral response was triggered in an all-or-none fashion. Pearson et al. (1979) used fathead minnows in an test of a assay system for complex industrial mixtures. They determined an LC_{50} for TNT of 2.4 mg/L in a 96-hour static test (Table 8.62). The range of LC_{50} values for 96-hour exposures to P. promelas was 1.2 to 2.58 mg/L.

One other fish species was tested for toxic effects by Klausmeier et al. (1982). They used the green swordtail Xiphorus helleri a tropical water species, and determined a LC_{50} of <5 mg/L in a 116-hour semistatic test (Table 8.62). They also noted behavioral patterns similar to those detailed by Smock et al. (1976) for the fathead minnow.

In addition to testing TNT solutions, several studies reported on the toxicity of by-products or wastewater mixtures containing TNT. Liu et al. (1976) tested 2,4-DNT on fathead minnows and determined LC_{50} values of 33.0 mg/L in a 24-hour static test and 31.0 mg/L in a 96-hour static test. Pearson et al. (1979) tested several TNT associated compounds using 96-hour static exposures to fathead minnows. They found LC_{50} values of 1.9 mg/L for 2,3-DNT, 32.5 mg/L for 2,4-DNT, and 0.12 mg/L for 2,3,6-TNT. Values for other by-products are given in Table 8.61 (Pearson et al. 1979). Another by-product associated with TNT, 1,3-dinitrobenzene, was tested for acute toxic effects in the fathead minnow by Curtis and Ward (1981). They used static 96-hour assays with a water temperature of 22°C, pH of 7.2-7.9, and water hardness of 40-48 mg/L (as $CaCO_3$) and determined an LC_{50} value of 12.7 mg/L.

An evaluation of the long-term toxicity of 2,4-DNT to bluegills was performed by Hartley (1981). He exposed juvenile bluegill to concentrations of 2,4-DNT ranging from 0.05 to 8.0 mg/L for eight weeks and observed changes in growth rates and histology of digestive tract, pancreas, skin, heart, gonads, kidney, and spinal cord. Growth rates were reduced, as indicated by first and second order growth constants, with increasing 2,4-DNT concentrations. The threshold level for growth effects was determined to be 0.05 mg/L. Histological abnormalities were found in the liver, spleen, trunk kidney, lateral line, and gills of fish exposed to 0.5 mg/L or greater concentrations. The effects

included hepatic lipid accumulation, hepatic necrotic foci, atypical renal tubules associated with tubule necrosis, atypical neuromast cells with necrotic epithelium in the lateral line mechanoreceptors, and hypertrophy of gill lamellae.

Evidence of toxicity of a TNT-mixture to fish was found in one study of the wastewaters from TNT manufacturing (Smock et al. 1976). This study used samples of synthetic mixtures designed to simulate the wastewater found in manufacturing processes. They prepared a synthetic wastewater mixture by exposing TNT to photochemical degradation and then tested fathead minnows in 96-hour static tests. They determined an LC_{50} value of 1.60 mg/L, which indicates a greater toxicity for the mixture than for the pure TNT solution that they tested.

8.5.1.5 Terrestrial Plants

Information on effects of TNT on terrestrial plants was not found in the literature. Data on acute exposures to three air pollutants associated with the manufacture of TNT were, however, found in Thompson and Kats (1978). The three compounds are sulfur dioxide, tetranitromethane, and mixed isomers of nitrotoluene. These compounds were tested individually on one-year-old seedlings of white oak (Quercus alba) and scotch pine (Pinus sylvestris) and on recently germinated seedlings (30 to 55 cm in height) of alfalfa (Medicago sativa), corn (Zea mays), wheat (Triticum aestivum), soybean (Glycine max), and tobacco (Nicotiana tobaccum). The plants were placed in a special fumigant chamber for 120 minutes and exposed to heat-vaporized concentrations of the test compounds at a flow rate of 7.4 m^3/min. Prior to exposure, the plants were kept in greenhouses with regulated temperature (18 to 32°C) and humidity (50 to 90 percent). For the first 72 hours after exposure, the plants were observed for damage to leaf structure. An EC_{50} value was calculated for the exposures based on damage to 50 percent of the leaves of all plants. The significant results are given in Table 8.63. The mixed isomers of nitrotoluene did not demonstrate any significant toxicity in any of the species when tested at 50 and 100 mg/m^3. Mature leaves of the two tree species did not demonstrate any significant toxic response to the two other TNT-associated compounds, but young leaves occasionally did react. Trinitromethane was the most toxic compound tested; the EC_{50} values were generally an order of magnitude lower than those of other compounds. The injury pattern for trinitromethane began after 24 hours with a grey stippling of leaves and developed into large yellow spots. Sulfur dioxide reacted similarly, but with more intensity. The reaction pattern consisted of bleaching of color and general wilting.

Combinations of sulfur dioxide, acetic acid, and trinitromethane were also tested for toxic effects on the two most sensitive plants, wheat and alfalfa (Thompson and Kats 1978). The effect criterion for this test was percentage of total leaf surface affected. The combination of sulfur dioxide and trinitrotoluene showed an antagonistic response when exposed to wheat, but not when the test plant was alfalfa. The nature of the response pattern was more typical of sulfur dioxide than of trinitromethane. The combination of acetic acid and sulfur

TABLE 8.63. COMPARATIVE TOXICITY OF TRINITROTOLUENE-ASSOCIATED COMPOUNDS TO DIFFERENT PLANT SPECIES AFTER 120-MIN EXPOSURES[a]

Species	Acetic Acid			Sulfur Dioxide			Trinitromethane		
	Number of Plants	EC_{50}[b] (mg/m^3)	EC_{50}[c] (mg/m^3)	Number of Plants	EC_{50}[b] (mg/m^3)	EC_{50}[c] (mg/m^3)	Number of Plants	EC_{50}[b] (mg/m^3)	EC_{50}[c] (mg/m^3)
Wheat	223	23.3	4.7-48.0	187	3.35	0-14.6	326	0.68	0-3.7
Alfalfa	110	7.8	5.8-10.2	228	6.3	3.6-47.6	285	0.93	0-4.77
Tobacco	239	41.2	4.5-79.4	323	18.6	0-132.4	187	6.1	1.14-12.2
Soybean	264	20.1	12.1-28.2	312	6.8	0-14.0	297	0.69	0-4.31
Corn	468	50.1	35.1-65.4	495	21.4	14.3-29.2	403	2.1	0.58-3.61

a. Adapted from Thompson and Kats 1978.

b. EC_{50} = concentration of phytotoxicant required to cause visible injury in 50% of the leaves of the exposed plant population.

c. 95% Confidence interval of concentrations in which true EC_{50} is expected. Zero values indicate that the statistical range includes negative values. Note: conversions to 1 ppm are: Acetic acid, 2.54 mg/m^3; sulfur dioxide, 2.61 mg/m^3; trinitromethane, 8.03 mg/m^3.

dioxide had no synergistic effects on wheat but did show a small antagonistic pattern in alfalfa. Again the response pattern was more typically that of sulfur dioxide than that of acetic acid.

These studies by Thompson and Kats (1978) indicate that the isomers of nitrotoluene are only a small phytotoxic hazard. The impact of sulfur dioxide is consistent with other information in the literature (Thompson et al. 1979). Trinitromethane was indicated as the most toxic waste compound associated with TNT manufacture.

8.5.1.6 Terrestrial Invertebrates

No data were found on the toxic effects of TNT on terrestrial invertebrates, either in laboratory tests or in field situations.

8.5.1.7 Birds

No data were found for the toxic effects of TNT on wild bird populations. One compound associated with TNT manufacture and degradation, 1,3-dinitrobenzene, was tested by Schafer (1972) in a large screen of chemical toxicity to wild birds. He tested oral toxicity (by gavage) in the starling (Sturnus vulgaris) and the redwing blackbird (Agelaius phoeniceus) and calculated LD_{50} values. For 1,3-dinitrobenzene, he determined an LD_{50} of >100 mg/kg in the starling and 42 mg/kg in the redwing. Of 61 compounds that he studied, the average LD_{50} values were 48 mg/kg for the starling and 15 mg/kg for the redwing.

8.5.1.8 Mammals

No data were found on the toxic effects of TNT on feral or domesticated mammals, either in laboratory studies or in field situations. Data on mammals frequently used as laboratory test animals are given in Section 7.1.2.

8.5.2 Toxic Effects of RDX and Associated Compounds on Organisms

Information on the toxic effects of the explosive compound RDX is more limited than information on TNT and its by-products. Thus many of the categories outlined below will lack any data and will be used to identify areas in need of more study.

8.5.2.1 Bacteria

No data were found on the toxic effects of RDX on bacteria, either in cultures or in field situations.

8.5.2.2 Aquatic Algae and Plants

In a comprehensive study of the aquatic toxicity of RDX, Bentley et al. (1977a) studied two blue-green algae species, Microcystsis aeruginosa and Anabeana flos-aquae, a green alga species, Selenastrum capricornutum, and a diatom species, Navicula pelliculosa. They exposed the

four species for 96 hours in a static test to nominal concentrations of RDX ranging from 0.32 to 32.0 mg/L and evaluated changes in numbers of cells and in chlorophyll a content. The four species all showed decreases in cell density at the 32 and 10 mg/L concentrations. RDX had the most effect on S. capricornutum, with a decrease of 38 percent in comparison with controls at the high dose and decreases from 2 to 23 percent at all dose levels. A similar pattern was shown for chorophyll a content, with all species affected at concentrations of 1.0 mg/L and higher, and S. capricornutum affected at all levels. The decreases ranged from 1 to 23 percent and were greater at higher concentrations. Bentley et al. (1977a) used probit analysis on the data and found the changes nonsignificant. However, in a review of RDX toxicity, Sullivan et al. (1979) applied additional statistical treatments to the data and found some statistical significance to the decreases. These were most significant for S. capricornutum, even at the lowest level. Sullivan et al. did not, however, equate the statistical significance with a biological significance at the lowest level. The no-effect-levels for cell density in the four species are: 10 ppm for M. aeruginosa, 0.32 ppm for A. flos-aquae, <0.32 ppm for S. capricornutum, and 3.2 ppm for N. pelliculosa (Sullivan et al. 1979).

In a field survey of the aquatic systems impacted by the Milan AAP, Huff et al. (1975d) were unable to attribute any significant effect on the phytoplankton and periphyton populations to the RDX concentrations in the water. This was due partly to poor habitat and interference from high water conditions. No other field data were found for RDX.

8.5.2.3 Aquatic Invertebrates

Bentley et al. (1977a) also tested four invertebrate species for acute toxic effects from RDX. They used the waterflea Daphnia magna, the isopod Asellus militaris, the amphipod Gammarus fasciatus, and the midge larvae Chironomus tentans in static tests of 24- and 48-hour duration at 20°C and 35 ppm hardness. The EC_{50} values, based on immobilization, were determined to be >100 mg/L for all species over both durations. A dynamic acute assay was also performed using the midge and waterflea species. The flow rate in these assays was 4 L/day and the other test variables were similar to those in the static tests. No effects were found in these tests, with EC_{50} values of >15 mg/L (the highest nominal concentration used).

In a field survey of the aquatic systems impacted by the Milan AAP, Huff et al. (1975d) were unable to attribute any significant effect on the zooplankton and macroinvertebrate populations to the RDX concentrations in the water. This was due partly to poor habitat and interference from high water conditions. No other field data were found for RDX.

8.5.2.4 Fish

The toxicity of RDX to four species of fish was determined by Bentley et al. (1977a) in static acute tests of 24-, 48-, and 96-hour durations. They exposed bluegills (Lepomis macrochirus), fathead minnows

(Pimephales promelas), rainbow trout (Salmo gairdneri), and channel catfish (Ictalurus punctatus) to RDX and found that the LC_{50} values ranged from 4.1 to 14 mg/L (Table 8.64). The channel catfish were the most sensitive of the species tested. A dynamic assay was performed using bluegills, rainbow trout, and channel catfish with a flow rate of 5 L/hour and temperature of 21°C. The LC_{50} values from these tests were in the same range as those of the static tests, except for the 96-hour value for the channel catfish, which was approximately 2.5 times greater (Table 8.65). They also tested various life stages of P. pimephales for acute toxicity using the static test conditions. They found that the 7-day posthatching stage was more sensitive than other stages, with LC_{50} values as low as 3.8 mg/L after 96 hours. The other stages showed widely varying LC_{50} values (Table 8.66). To verify that test conditions were not a significant factor, they varied the temperatures from 15 to 25°C, the pH from 6 to 8, and the hardness (in mg/L $CaCO_3$) from 35 to 250 in a static test using L. macrochirus. The LC_{50} values were generally equivalent with some decrease at lower temperatures (Table 8.67). A similar test of the effect of aging the test RDX solution produced identical LC_{50} values, indicating that little significant effect can be attributed to the test conditions.

An evaluation of the toxic effects on P. promelas from exposure to wastewater from an AAF producing HMX and RDX explosives was performed by Stilwell et al. (1977). They used various dilutions of the waste effluents that contained the explosives at levels no greater than 6.0 ppm and exposed the fish for 96 hours in a static assay. The LC_{50}

TABLE 8.64. ACUTE TOXICITY VALUES FOR RDX[a] IN FISHES DETERMINED DURING STATIC TOXICITY TESTS[b]

Species	LC_{50} (mg/L)		
	24-hr	48-hr	96-hr
Lepomis macrochirus (bluegill)	14 (12-17)[c]	8.5 (7.5-9.5)	6.0 (5.-6.5)
Salmo gairdneri (rainbow trout)	9.4 (8.5-10)	7.0 (6.3-7.7)	6.4 (5.4-7.4)
Ictalurus punctatus (channel catfish)	7.5 (0.7-8.5)	6.0 (5.3-6.9)	4.1 (3.5-4.9)
Pimephales promelas (fathead minnow)	10 (7.4-14)	5.8 (6.9-12)	5.8 (4.7-7.2)

a. RDX = hexahydro-1,3,5-trinitro-1,3,5-triazine.

b. Adapted from Bentley et al. 1977a.

c. 95% Confidence interval.

TABLE 8.65. ACUTE TOXICITY OF RDX[a] TO FISHES DURING DYNAMIC TOXICITY TESTS (NOMINAL CONCENTRATION)[b]

Species	LC_{50} (mg/L)		
	24-hr	96-hr	Incipient[c]
Lepomis macrochirus (bluegill)	>10	7.6 (5.6-10)[d]	6.4 (5.3-7.8)
Ictalurus punctatus (channel catfish)	>10	13 (8.8-20)	11 (9.1-13)
Pimephales promelas (fathead minnow)	>10	6.6 (5.0-8.7)	5.2 (4.3-6.4)

a. RDX = hexahydro-1,3,5-trinitro-1,3,5-triazine.

b. Adapted from Bentley et al. 1977a.

c. Incipient LC_{50} estimated after 264 hours.

d. 95% Confidence interval.

TABLE 8.66. ACUTE TOXICITY OF RDX[a] TO SELECTED LIFE STAGES OF FATHEAD MINNOWS (Pimephales promelas) AS DETERMINED DURING STATIC TOXICITY TESTS[b]

Life Stage	LC_{50} (mg/L)			
	24-hr	48-hr	96-hr	144-hr
Eggs	>100	>100	>100	>100
1-hour Post hatch	>100	>100	43 (27-69)[d]	-[c]
7-day Post hatch	32	18 (13-24)	3.8 (3.0-5.0)	-
30-day Post hatch	18 (13-24)	16 (13-19)	16 (13-19)	-
60-day Post hatch	11 (6.1-21)	11 (5.9-21)	11 (5.9-21)	-

a. RDX = hexahydro-1,3,5-trinitro-1,3,5-triazine.

b. Adapted from Bentley et al. 1977a.

c. Tests with fry were 96 hours in duration.

d. 95% Confidence interval.

TABLE 8.67. ACUTE TOXICITY OF RDX[a] TO BLUEGILL (Lepomis macrochirus) UNDER VARYING CONDITIONS OF WATER QUALITY DURING STATIC TOXICITY TESTS[b]

Temperature (°C)	pH	Hardness (mg/L $CaCO_3$)	96-hr LC_{50} (mg/L)
15	7.0	35	8.4 (6.0-11)[c]
20	7.0	35	5.1 (3.9-6.7)
25	7.0	35	4.1 (3.0-5.6)
20	7.0	35	3.8 (2.0-7.1)
20	7.0	100	5.3 (4.1-5.8)
20	7.0	250	3.9 (2.1-7.3)
20	6.0	35	3.6 (1.9-6.6)
20	7.0	35	3.7 (2.0-6.9)
20	8.0	35	3.9 (2.1-7.3)

a. RDX = hexahydro-1,3,5-trinitro-1,3,5-triazine.

b. Adapted from Bentley et al. 1977a.

c. 95% Confidence interval.

values generated depended on the part of the plant from which the effluent was taken and ranged from 1 percent to 70 percent of the fluent concentrations. Some of the solutions failed to produce any lethality. Correlation coefficients were calculated and indicated no positive correlation between the LC_{50} values and the RDX content of the water when the controlled variable was biological oxygen demand or COD. However, when ammonia or total nitrogen were the controlled variables, the correlation between RDX and toxicity was significant.

In a field survey of the aquatic systems impacted by the Milan AAP, Huff et al. (1975d) were unable to attribute any significant effect on the fish populations to the RDX concentrations in the water. This was due partly to poor habitat and interference from high water conditions. No other field data were found for RDX.

8.5.2.5 Terrestrial Plants

No data were found on the toxic effects of RDX to terrestrial plants, but data were found for some compounds associated with manufacture or degradation of RDX. Thompson and Kats (1978) tested acetic acid, sulfur dioxide, nitromethane, and methyl nitrate for acute toxic effects on white oak (Quercus alba), scotch pine (Pinus sylvestris), alfalfa (Medicago sativa), corn (Zea mays), wheat (Triticum aestivum), soybean (Glycine max), and tobacco (Nicotiana tobaccum). The trees were tested as one-year-old seedlings and the others as recently germinated (30 to 55 cm in height) plants. The plants were placed in a special fumigant chamber for 120 minutes and exposed to heat-vaporized concentrations of the test compounds at a flow rate of 7.4 m^3/min. Prior to

exposure, the plants were kept in greenhouses with regulated temperature (18 to 32°C) and humidity (50 to 90 percent). For the first 72 hours after exposure, the plants were observed for damage to leaf structure. An EC_{50} value was calculated for the exposures based on damage to 50 percent of the leaves of all plants. The data on sulfur dioxide are given in Table 8.63 and discussed in the TNT Section 8.5.1.5, because sulfur dioxide is also associated with TNT production. Acetic acid produced a bleaching of color and general wilting. Nitromethane failed to show any toxic effects on the plants, even at 50 mg/m^3, the highest concentration used. Methyl nitrate caused only slight injury to wheat and moderate injury to alfalfa at 20 mg/m^3. No effects were seen in the other plants at twice this concentration. Tests of alfalfa and wheat at this higher dose produced only slightly more injuries than the lower dose, and the EC_{50} values would have been in excess of this. Consequently, the authors stopped testing methyl nitrate at this point. The pattern of effects for wheat consisted of distal lesions and, for alfalfa, of necrotic spots on the margins.

8.5.2.6 Terrestrial Invertebrates

No data were found on the toxic effects of RDX on terrestrial invertebrates, either in laboratory tests or in field situations.

8.5.2.7 Birds

No data were found on the toxic effects of RDX on wild bird populations, either in laboratory tests or in field situations.

8.5.2.8 Mammals

No data were found on the toxic effects of RDX on feral or domesticated mammals, either in laboratory studies or in field situations. Data on mammals frequently used as laboratory test animals are given in Section 7.2.2.

8.5.3 Toxic Effects of HMX and Associated Compounds on Organisms

Information on the toxic effects of the explosive compound HMX is even more limited than information on RDX and its by-products. Thus, many of the categories outlined below will lack any data and will be used to identify areas in need of more study.

8.5.3.1 Bacteria

No data were found on the toxic effects of HMX on bacteria, either in cultures or in field situations.

8.5.3.2 Aquatic Algae and Vascular Plants

In a comprehensive study of the aquatic toxicity of HMX, Bentley et al. (1977b) studied two blue-green algae species, Microcystsis aeruginosa and Anabeana flos-aquae, a green algae species, Selenastrum capricornutum, and a diatom species, Navicula pelliculosa. They exposed the

four species for 96 hours in a static test to concentrations of HMX ranging from 0.32 to 32.0 mg/L and evaluated changes in numbers of cells and in chlorophyll a content. At none of the concentrations were any deleterious effects found; in comparisons (using probit transformations) with control values the two parameters showed no significant increases, and the EC_{50} level must be considered in excess of 32 mg/L. In a review of HMX toxicity, Sullivan et al. (1979) applied additional statistical treatments to the data and found some statistical significance to the increases. However, they did not equate the statistical significance with a biological significance. No data were found for effects of HMX on vascular aquatic plants.

8.5.3.3 Aquatic Invertebrates

Bentley et al. (1977b) also tested four invertebrate species for acute toxic effects from HMX. They used the waterflea Daphnia magna, the isopod Asellus militaris, the amphipod Gammarus fasciatus, and the midge larvae Chironomus tentans in static tests of 24- and 48-hour durations at 20°C and 35 ppm hardness. The EC_{50} values, based on immobilization, were all in excess of the highest level used, 32 mg/L.

8.5.3.4 Fish

The toxicity of HMX to four species of fish was determined by Bentley et al. (1977b) in static acute tests of 24-, 48-, and 96-hour durations. They exposed bluegills (Lepomis macrochirus), fathead minnows (Pimephales promelas), rainbow trout (Salmo gairdneri), and channel catfish (Ictalurus punctatus) to HMX and found that the LC_{50} values for all cases was >32 mg/L. They also tested various life stages of P. pimephales for acute toxicity using the same test conditions. They found that the 7-day posthatching stage was more sensitive than other stages, with LC_{50} values as low as 15 mg/L after 96 hours (Table 8.68). To verify that test conditions were not a significant factor, they varied the temperatures from 15 to 25°C, the pH from 6 to 8, and the hardness (in mg/L $CaCO_3$) from 35 to 250 in a static test using L. macrochirus. The LC_{50} values were all >32 mg/L, indicating that no significant effect can be attributed to the test conditions.

An evaluation of the toxic effects on P. promelas from exposure to wastewater from an AAP producing HMX and RDX explosives was performed by Stilwell et al. (1977). They used various dilutions of the waste effluents that contained the explosives at levels no greater than 6.0 ppm and exposed the fish for 96 hours in a static assay. The LC_{50} values generated depended on the part of the plant from which the effluent was taken and ranged from 1 percent to 70 percent of the effluent concentrations. Some of the solutions failed to produce any lethality. Correlation coefficients were calculated and indicated a positive correlation between the LC_{50} values and the HMX content of the water.

TABLE 8.68. ACUTE TOXICITY OF HMX[a] TO SELECTED LIFE STAGES OF FATHEAD MINNOWS (Pimephales promelas) AS DETERMINED DURING STATIC BIOASSAYS[b]

Life Stage	LC_{50} (mg/L)			
	24-hr	48-hr	96-hr	144-hr
Eggs	>32	>32	>32	>32
1-hr Post hatch	>32	>32	>32	-[c]
7-day Post hatch	>32	25 (7.6-81)[d]	15 (8.8-26)	-
30-day Post hatch	>32	>32	>32	-
60-day Post hatch	>32	>32	>32	-

a. HMX = 1,3,5,7-tetranitro-1,3,5,7-tetrazacyclooctane.

b. Adapted from Bentley et al. 1977b.

c. Egg testing conducted for 144 hours; all other life stages conducted for 96 hours.

d. 95% Confidence interval.

8.5.3.5 Terrestrial Plants

No data were found on the toxic effects of HMX on terrestrial plants, either in laboratory tests or in field situations.

8.5.3.6 Terrestrial Invertebrates

No data were found on the toxic effects of HMX on terrestrial invertebrates, either in laboratory tests or in field situations.

8.5.3.7 Birds

No data were found on the toxic effects of HMX on wild bird populations, either in laboratory tests or in field situations.

8.5.3.8 Mammals

No data were found on the toxic effects of HMX on feral or domesticated mammals, either in laboratory studies or in field situations. Data on mammals frequently used as laboratory test animals are given in Section 7.3.2.

8.6 CONCLUSIONS

In reviewing the information on environmental effects of TNT, RDX, HMX, and associated pollutants, an impression is gained that, although the short-term impact has been minimal, the potential for a greater long-term impact exists. Due to this potential, research in several areas is needed to complete our understanding of the environmental impacts of these munitions.

8.6.1 Impacts on Aquatic Systems

Data on releases from AAPs indicate that aquatic systems receive the majority of the wastes. The effluents as they leave the plants have a significant potential for harmful effects. Levels of pollutants have been reported from TNT manufacturing areas with ranges of 0.05 to 178 mg/L for TNT, 2.4 to 200 mg/L for nitrogen compounds, 1.53 to 1,000 mg/L for sulfates, and 130 to 2,000 mg/L for dissolved solids. At RDX/HMX sites, effluent levels of 0.1 to 109 mg/L for RDX and 0.09 to 3.36 mg/L for HMX have also been reported. Although levels measured in the environment are much lower (for TNT, up to 1,020 µg/L in water and up to 18.7 mg/kg in sediment; for RDX up to 110 µg/L in water and up to 43 mg/kg in sediment), the potential for hazardous effects exists.

Studies on the fate and movement of TNT, RDX, and HMX in aquatic systems indicate that long distance transportation (>20 km) of the munitions and their degradation products is not highly probable. Photolytic degradation of the compounds proceeds fairly rapidly and limits the persistence of the munitions in the water column. However, microbial degradation does not proceed rapidly, especially for RDX, and longer persistence in sediments is a distinct possibility. This possibility is further indicated by higher levels in samples of sediments vs water.

Studies on bioaccumulation indicate that this is not a problem for the munition compounds of concern.

The impact of the AAP effluents on the water quality has been demonstrated. Various measures of the habitat quality of water and sediments are changed including chemical oxygen demand; concentrations of solids, nitrogen-species, phosphates, and sulfates; and pH levels. These effects are usually restricted to areas immediately downstream from the AAPs, but may be quite persistent in sediments.

Studies of the toxic effects of TNT to aquatic organisms indicate a potential for problems in the environment. Levels found to be toxic (~100 ppb to 1 ppm) to bacteria, plants, and invertebrates in terms of growth suppression or habitat utilization have been reported for both water and sediment. Levels that actually exceed LC_{50} values have also been reported for some sediment samples and for effluents from the AAPs and indicate a further hazard potential for species of bacteria, algae, vascular plants, invertebrates, and fish. For example, the 18.7-ppm sediment level of TNT exceeds published LC_{50} ranges of 5-10 ppm for aquatic invertebrates. This potential extends to degradation products

and impurities associated with TNT production (e.g., 2,4-DNT and 2,3,6-TNT). Potential toxic effects of RDX should be less of a concern because the effective levels are higher than those of TNT. This trend continues to a greater degree for HMX.

8.6.2 Atmospheric Impacts

The potential for deleterious effects from atmospheric releases of TNT, RDX, HMX, and associated compounds is related to the various smaller constituent compounds arising from the manufacture of the munitions. The principal species of concern are sulfur dioxide, nitrogen oxides, and particulates. These have been found to exceed the allowable EPA levels in releases from AAPs in terms of both daily and yearly amounts. The small amount of information available on transportation and persistence suggests that the problems associated with the releases should be local in nature. Studies of the toxic effects of these releases are lacking, but the information available indicates that the sulfur dioxide releases pose a potential problem for surrounding vegetation.

8.6.3 Terrestrial Impacts

Data on the impact of TNT, HMX, and RDX on the soils, groundwater, and associated terrestrial ecosytems are generally insufficient for in-depth analysis. Data available on the input of munitions to terrestrial systems suggest that high levels could be obtained in limited areas. Since other information indicates that the munitions may be persistent at depths due to slow biodegradation, some concern is merited regarding potential deleterious effects. Migration studies do indicate that the munitions move slowly through soils and are found only at low levels in the groundwater, which should limit the immediate impact to local areas. Chronic groundwater contamination is an associated potential hazard from terrestrial releases. The most likely source for problems of these types would be improper disposal in landfills.

8.6.4 Data Gaps and Research Recommendations

The following list details some of the areas in which additional research would aid in evaluating the true hazard posed to the environment by the release of waste products from AAPs and munition manufacture.

1. The deposition of released wastes into aquatic sediments and the associated interaction between levels in sediments and in water.

2. The persistence of deposited wastes in sediments from various aquatic systems.

3. A more detailed evaluation of the effects (including chronic) of released wastes and individual munitions on the aquatic benthic communities and principal invertebrate species (aquatic insects).

4. Chronic exposure studies evaluating toxicity of munitions and wastes to fish.

5. Toxicity studies on the effects of individual munitions and waste mixtures on aquatic vascular plants.

6. Toxicity studies on RDX and HMX exposures to bacteria species.

7. More detailed studies of the degradation pathways of TNT, RDX, and HMX in aquatic and terrestrial systems, in particular to determine if ring structure of the munitions is broken.

8. Additional toxicity studies (including chronic studies) on the effects of munition exposures on organisms in terrestrial systems including plant, mammal, insect, decomposer, and bird species.

9. In conjunction with recommendation 7, on-site evaluations of effects of released wastes, particularly effects of atmospheric releases on surrounding vegetation.

10. Identification of potential for long-term transport in soils and groundwaters of AAP sites.

11. Determination of long-term persistence of munitions and wastes in soil systems at depths below the photolytic zone.

12. Evaluation of the particulates released atmospherically from AAPs to determine munitions content, transport, persistence, and degradation.

13. Determination of the effectiveness of pollution abatement and treatment methodologies in limiting the environmental entry of waste products from AAP and LAP facilities.

14. Determination of the rates of subsurface transport of wastes into groundwater because low sorption and long biological half-lives suggest that this may be a significant problem.

9. MUNITION PLANT WASTE TREATMENT

9.1 INTRODUCTION

Army Ammunition Plants (AAPs) were operated during the emergency situation of World War II with minor concern for the environment (Ziegler 1980). Public awareness of environmental pollution during the late 1960s resulted in Executive Order 11597, dated February 1970, and in later superseding orders that required all federal facilities to conform to federal environmental regulations. The Army then embarked on a program of intensive modernization and expansion in 1970. The first order of priority in the modernization program was assigned to the propellants and explosives production facilities because these were the major sources of pollution. During the period 1970-77, over 27 million dollars was budgeted for this purpose. Since then, 1-2 million dollars have been budgeted annually for the environmental technology program. During the early phase, the major thrust was to assess known technologies and to correct known deficiencies. Currently, the major thrust has shifted to fine-tuning existing technologies to make them more cost effective, locating new and superior technologies to reduce operating costs, and complying with new, more stringent federal, state, and local requirements for control of pollutants.

Each munition plant is subjected not only to the U.S. Environmental Protection Agency (EPA) regulations but also to state and local regulations (Ziegler 1980). This necessitates a customized approach for each plant to comply with the various standards at an economical cost.

Potential waste handling problems associated with the production of TNT, RDX, and HMX in AAPs are briefly discussed below in three sections: (1) atmospheric emissions, (2) aqueous effluents, and (3) solid wastes.

9.2 ATMOSPHERIC EMISSIONS

Four major air pollutants generated during the manufacture of TNT are nitrogen oxides produced during the nitration step in the manufacture of TNT, tetranitromethane produced during the nitration of toluene to make TNT, mononitrotoluene emitted during the manufacture of TNT, and acid mist produced in the sulfuric acid recovery plant (Roth 1980; Ziegler 1980).

As discussed by Ziegler (1980), air pollution was a problem with the older batch process of manufacturing TNT. After the installation of new continuous TNT lines such as the continuous automated multibase line at Radford Army Ammunition Plant (RAAP), however, much of the air emission has been greatly reduced. In experimental tests, molecular sieve absorption has been found to be most effective for achieving the lowest level of NO_x. When NO_x emissions contain nitrobodies such as tetranitromethane, a two-stage scrubbing with sulfuric acid in conjunction with another two-stage scrubbing with sellite has been found to be satisfactory. Tetranitromethane can also be treated with sodium carbonate and stabilized hydrogen peroxide to form trinitromethane (Gilbert

1980). However, the effectiveness of the sellite absorption tower for absorption of tetranitromethane has been demonstrated only in pilot plant experiments, and, despite such a system being in place at RAAP, it is not currently in use because of difficulties in getting it to work on a large scale. Also, the molecular sieve has not used in connection with TNT manufacture at RAAP. The bottom line is that tetranitromethane is discharged unabated to the atmosphere during current TNT production at Radford (Eaton 1984).

Atmospheric emissions from RDX manufacture at Holston AAP have been surveyed and are included in Table 8.12. The major EPA criteria pollutants are particulates, sulfur oxides, carbon monoxide, and nitrogen oxides. The major organic pollutants are acetic acid, n-propylacetate, cyclohexanone, acetone, and methyl nitrate. Although no separate survey of the emissions from HMX manufacture was found, the pollutants are expected to be similar because both RDX and HMX are manufactured by modifications of the Bachmann process, although different reaction conditions are employed.

9.3 AQUEOUS EFFLUENTS

9.3.1 Red Water

A major source of water pollution is red water arising out of the manufacture of TNT. Its composition has been described in detail in Section 5.2.1. It contains TNT isomers, sodium carbonate, sodium sulfate, sodium sulfite, and other complex chemicals (Ziegler 1980). Many components in red water are toxic and carcinogenic. During World War II and the Korean War, red water was dumped into streams as a means of disposal. At one time red water, after concentration at the TNT plant, was sold to paper mills where it was incinerated to sodium sulfate for use in pulping. However, this avenue of disposal is no longer feasible because of the cost of incineration as well as the problems associated with transportation of red water since it has been classified as "hazardous substance" by EPA (Hall and Lawrence 1976, Ziegler 1980). The other alternative is incineration; but this is expensive and adds to air and solid waste pollution. The recovery of sulfur and sodium present in red water seems to be the best way to tackle this problem, according to Ziegler (1980). Several technologies are available and include: (1) Atomics International, a molten salt bath reduction process; (2) Tampella Smelt, a carbonate process; (3) SCA Billerud, a pyrolysis reduction process; and (4) SONOCO, a sulfite recovery process.

Based on the capital and operating costs, ease of operation, simplicity of design, and technical aspects, the SONOCO sulfite recovery process appears attractive. In the sulfite recovery process adapted for Radford AAP, red water is mixed with aluminum hydroxide and reduced in a furnace to sulfur dioxide and soluble sodium aluminate. The sulfur dioxide is scrubbed with sodium carbonate to form sodium sulfite, which is reused to purify TNT. The sodium aluminate is converted to aluminum hydroxide, which is recycled for the treatment of the red water feedstock. The current status of this process has not been reported in the literature, but it was projected to be in operation in FY 82 (Eckenrode et al. 1980).

The feasibility of recovering the major organic components (2,4-dinitrotoluene-3-sulfonate, 2.0 percent and 2,4-dinitrotoluene-5-sulfonate, 2.7 percent) in red water has been investigated by Gilbert (1977) and Hall and Lawrence (1976) as a means to economize the processing of red water. It is possible to recover these materials from red water and convert them into useful products such as 2,4-dinitrocresol.

9.3.2 Pink Water

The composition and sources of pink water have been discussed in Section 5.2.2. Pink water is generated during TNT manufacture, LAP, and demilitarization operations. It contains varying amounts of α-TNT, meta TNT isomers, and RDX depending on the source (see Table 5.2). According to Forsten (1980), the volume of pink water at full mobilization may be as high as 100,000 gallons/day per line and may contain 140-160 mg TNT/L and 85-90 mg RDX/L. At present activated charcoal is widely used for pink water abatement, with a cost of $7.43 per 1,000 gallons treated. A new thermal carbon regeneration process using rotary kilns has been developed and has lowered the cost to $3.48 per 1,000 gallons (Ziegler 1980). In this process, the explosive-laden carbon is first dewatered and calcined at 110°C (Forsten 1980). In the second step, spent carbon is pyrolyzed at 300°C to remove the adsorbed explosives. Steam at 20 kg/hr and carbon dioxide at 25 L/min are fed at this stage to maintain a reducing atmosphere. In the third step, the carbon is subjected to a reactivation temperature of 861°C. A regeneration efficiency of 92 percent has been achieved (Forsten 1980).

9.4 SOLID WASTES

In the manufacturing and LAP operations involving munitions, various unusable solid wastes are generated and must be disposed of in an ecologically acceptable manner (Zeigler 1980). Federal and state regulations must also be considered when selecting effective waste disposal methodologies. As discussed by Zeigler (1980), controlled incineration is required, and the methodologies that have been investigated include the fluidized bed, rotary kiln, and SITPA II (Simplified Incinerator Technology for Pollution Abatement) systems.

9.5 CONCLUSIONS

In modernized AAPs, air pollution will apparently not be a significant problem, and plans call for controlled incineration of solid explosive containing wastes. Nitrogen oxides and tetranitromethane can be effectively removed by a two-stage scrubbing with sulfuric acid in conjunction with another two-stage scrubbing with sellite.

Red water and pink water are the major sources of potential water pollution. The SONOCO sulfite recovery process for the treatment of red water seems very promising. Pink water is treated by activated charcoal. A method has been developed for the regeneration of the explosive-laden charcoal with 92 percent efficiency, thus considerably reducing the operating costs.

10. GLOSSARY

AAP - army ammunition plant.

ACGIH - American Conference of Governmental Industrial Hygienists.

Acute toxicity test - test to determine the level at which a single dose of a toxicant shows a specific adverse effect on a specified percentage of homogeneous test organisms in a specific short period of time.

ADNT - aminodinitrotoluene.

alpha-TNT - 2,4,6-trinitrotoluene, α-isomer.

AOP - ammonia oxidation plant.

APSA - Ammunition Procurement and Supply Agency, U.S. Army.

Bachmann process - a process for the manufacture of RDX and HMX (by-product) from hexamine using ammonium nitrate and acetic anhydride in addition to nitric acid.

Bioaccumulation - the process whereby certain toxic substances collect in living tissue; may pose substantial hazard to human health or environment.

BOD - biological oxygen demand.

BUN - blood urea nitrogen.

CAS - Chemical Abstract Service.

Chronic toxicity test - test to determine the effects of a toxicant administered regularly over a long period of time.

Compound B - munition mixture containing 60 percent RDX and 40 percent TNT.

COD - chemical oxygen demand.

cP - centipoise; one hundredth of a poise (centimeter-gram-second unit of dynamic viscosity).

DNT - dinitrotoluene.

EC_{50} - median effective concentration; generally a concentration at which 50 percent of the test organisms respond in the measured parameter (e.g., react in a specified behavorial pattern).

EPA - Environmental Protection Agency.

GOCO - government owned and contractor operated.

GOGO - government owned and government operated.

Half-life - the length of time required to reduce the concentration of a compound by half; often refers to radioactive decay or elimination of a compound from an animal.

Hercules process - continuous method of manufacture of TNT from toluene and H_2SO_4-HNO_3.

HMX - octahydro-1,3,5,7-tetranitro-1,3,5,7-tetrazocine; cyclotetramethylene tetranitramine.

IR - infrared.

J/g - joules/gram.

J/(gK) - joules/(gram, Kelvin).

kJ/g - kilojoules/gram.

Landfill - disposal of waste by burying it under layers of earth material in low-lying ground.

LAP - load, assemble, and pack; operations involved in preparing munition products.

LAP(I) - wastewater from a load, assemble, and pack area that has been photolytically degraded.

LC_{50} - concentration of a test substance that is lethal to 50 percent of a group of test organisms; similar to LD_{50} but used in aquatic or inhalation toxicity tests.

LD_{50} - dose of a test substance that is lethal to 50 percent of a group of test organisms.

MNT - mononitrotoluene.

NPDES - National Pollutant Discharge Elimination System, Environmental Protection Agency.

OSHA - Occupational Safety and Health Administration.

Photodissociation - the removal of one or more atoms from a molecule by the absorption of a quantum of electromagnetic energy.

Photolysis - chemical decomposition induced by light or other radiant energy.

Pink water - aqueous effluents from plants that manufacture or process TNT.

PMR - proton magnetic resonance.

RDX - hexahydro-1,3,5-trinitro-1,3,5-triazine; cyclotrimethylene trinitramine.

Red water - the brick-red extract of crude TNT with sellite.

Red Tar - composition of organic sulfonates derived from 2,4,6-TNT as a result of overly drastic conditions of selliting.

ROF - royal ordnance factory.

SAC - sulfuric acid concentrator.

Sellite - sodium sulfite/sodium carbonate solution.

SEX - 1-acetyloctahydro-3,5,7-trinitro-1,3,5,7-tetrazocine.

SGOT - serum glutamic oxalacetic transaminase; an enzyme that catalyzes transfer of ammonia between certain organic compounds.

STEL - short-term exposure limit.

Subchronic toxicity test - mutiple dose toxicity test conducted for shorter time period than chronic test; often used as a preliminary evaluation of long-term effects.

TAX - 1-acetylhexahydro-3,5-dinitro-1,3,5-triazine.

TDS - total dissolved solids.

Tetryl - explosive used in detonators; tetranitromethylaniline.

TKN - total Kjedahl nitrogen.

TLC - thin-layer chromatography.

TLV - threshold limit value; the concentration of a substance in air considered acceptable for exposure of industrial workers.

TNM - tetranitromethane.

TNT - 2,4,6-trinitrotoluene; alpha-trinitrotoluene.

TOC - total organic carbon.

TWA - time weighted average.

μmhos/cm - microsiemens/cm; measure of electrical conductance.

W/(m.K) - watt/(meter Kelvin).

Woolwich process - a process of manufacture of RDX by direct nitrolysis of hexamine with nitric acid.

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